L. XCV JANUARY, 1932 Medical Lib. JAN 8 1932 No. 1

ANNALS of SURGERY

A Monthly Review of
Surgical Science and Practice

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Official Publication of the American Surgical Association, of the New York Surgical Society and the Philadelphia Academy of Surgery

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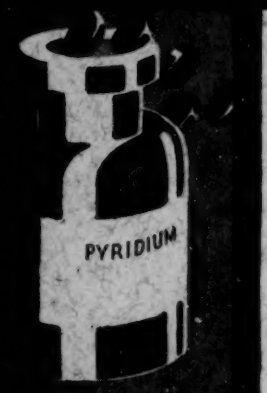

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ANNALS *of* SURGERY

Vol. XCV JANUARY, 1932 No. 1

CONGENITAL CYSTS AND FISTULÆ OF THE NECK*

By Herbert Willy Meyer, M.D.,

of New York, N. Y.

Early in the author's surgical life the opportunity of studying and operating upon a number of recurrent thyro-glossal fistulæ cases presented itself. These cases had been admitted to the service of Dr. George H. Semken, at the N. Y. Skin and Cancer Hospital. Naturally, interest in the cause of these conditions was aroused. Later, the occasion to operate upon a patient with a complete lateral fistula arose. In all the series of thyro-glossal fistulæ cases amounted to ten cases.

This experience stimulated a closer study of the underlying causes of these interesting and somewhat unusual conditions.

In studying the literature innumerable references were found and quite a difference of opinion as to the etiological factors. Some of the more important recent works in the English literature were those of Christopher, Klingenstein and Colp, and Semken's article in Nelson's Loose Leaf Surgery together with many others.

One of the most important single contributions from abroad appeared in 1908 and 1912 by Romuald Wenglowski, a Russian surgeon and investigator living in Moscow.

Upon closer study of his monograph we were greatly impressed by the thoroughness of his work and the correctness of his theories and therefore came to the conclusion to present his principles and theories in greater detail.

Having made these studies one can readily understand why so many of these patients have recurrences of the mid-line fistulæ after operation. It is for this reason that this paper will deal with the embryological factors underlying lateral and medial cysts and fistulæ of the neck, and with the resulting principles of thorough and radical primary operations based upon the etiological factors.

The paper will be divided into three parts, the first dealing with the lateral cysts and fistulæ, the second with the medial cysts and fistulæ, while the third division will be in concise form, dealing with the other congenital conditions that may be found in the neck.

PART I. LATERAL CYSTS AND FISTULÆ OF THE NECK

Historical Facts.—In 1832, Ascherson published an article in which he stated that these cysts and fistulæ were related to the branchial clefts, the higher the opening of the

* Read before the New York Surgical Society, October 28, 1931.

fistula in the neck, the higher the cleft involved in its formation. Heusinger stated that the opening was usually low, and that therefore the fourth cleft was more frequently involved than the second or third. Bland-Sutton made a chart showing which cleft was involved and that it depended upon the location of the opening as to the cleft involved.

The greatest work done on this subject appeared in the early 'eighties by His. He stated that fistulæ of the neck were a normal finding up to the end of the fifth embryonic week; further, that the sinus præcervicalis was the anlage for the thymus; and he mentioned the relationship between the thymus and neck fistulæ. The sinus præcervicalis, His stated, only rarely breaks through into the pharynx, but thereby he explained the inner opening of the neck fistulæ. The inner opening depends upon which of the clefts breaks through; if the second, then in the Rosenmüller groove in the supratonsillar fossa; if the third, then under the plica nervi laryngei; if the fourth, then in the pyriform sinus. In 1899, His changed his opinion when he found evidences that the thymus did not develop from the sinus præcervicalis, but from the third branchial cleft.

Rabl believed that the fistulæ were due to the perforation of a thin membrane between the sinus cervicalis and the pharynx. He called it the "Kiemengang." He felt that the third cleft was impossible as an etiological factor on account of the development of the thymus. Further, the fourth cleft was impossible on account of the thick mesoderm between it and the pharynx. Therefore, he believed that the second cleft was the one most responsible.

In 1890 Kostaniecki and Milecki made some very thorough literary studies. They did not do original work, but brought order out of the chaos of all previous publications in this involved subject. They believed that the outer opening had no relation to the number of the cleft involved. If the outer opening depended on the cleft number then the inner opening would have to also, and the literature did not show this to be the case. The inner opening was almost always in the same place, in the lower tonsillar fossa near the root of the tongue and more posterior. As this region is that of the second cleft they believed this to be the one at fault. The theory was that the second pharyngeal pouch broke through into the sinus cervicalis and then separated itself from the pharynx. Up to the work of Wenglowski this literary work, study and theory was the best published.

Wenglowski did not see the rationale of this theory. Virchow's description of a case in 1865, attributed by the others to the first cleft, actually was a coincidence of an abnormality of the outer and middle ear combined with a lateral fistula of the neck. Watson described a fistula which Kostaniecki and Milecki ascribed to the second cleft, but the tract passed under the stylopharyngeus muscle which develops in the third branchial arch. Therefore, the fistula also would have to be ascribed to the third cleft. Kostaniecki and Milecki themselves were annoyed by this fact, but they explained it by an abnormal course of the muscle fibres. If the fistula belongs only to the second cleft, then the inner opening would have to be strictly within the field of the second cleft. The boundaries are the tonsillar fossa, bounded in front by the palatoglossal arch and posteriorly by the palatopharyngeal arch. It has been shown that the inner opening is not in the middle of the tonsil nor between these arches. The majority of the cases described open below the tonsillar fossa next to the root of the tongue and behind the palatoglossal arch. Therefore, they have a much closer relation with the third cleft, as the palatoglossal arch and the underlying muscle belong to the third and not to the second arch. It is difficult to explain according to Kostaniecki and Milecki's theory why a complete fistula forms an arch, the upper segment of which runs behind and mesial to the angle of the jaw. It is also difficult to explain why the fistula curves downward and does not correspond to any other branchial groove, even if we believe that the second pouch had broken through into the sinus cervicalis, and both had remained open. They believed that the outer opening depends on the size and descent of the cysts and the area of perforation of the cysts. Upon careful study of clinical histories, however, it is quickly seen that there is a certain uniformity in the region where the outer openings occur. They open anywhere along the medial border of the sternomastoid muscle, from the angle of the jaw

down to the middle of the sternum. As further evidence against Kostaniecki and Milecki's theory is the fact that the entire branchial apparatus in the human being does not spread downward onto the neck, but the second, third and fourth branchial arches pass posteriorly. Within the second arch develops the hyoid bone, while out of the third and fourth arches come the cornu of the hyoid and some of the muscles that insert on the hyoid from above. Therefore, the boundary of the branchial apparatus must be above the line bounded below by the lower border of the hyoid. The sinus cervicalis together with the third and fourth arches lies within this field, high under the angle of the jaw. As the arches and pouches are always uniform in position anatomically and topographically, therefore the fistula, if caused by the process of the opening of the cleft through the fistulous opening, would have to be in the region of the angle of the jaw and the upper portion of the neck. However, most of the fistulæ occur in the middle or lower neck and only rarely run upward and end blindly at the angle of the jaw. The histological findings of mixed ciliated and squamous epithelium also speak against the branchiogenetic origin, which would have to have ciliated epithelium near the pharyngeal opening and squamous epithelium in the more distal parts. This could never be proven. For these reasons Wenglowski made investigations to try to find another causative factor which would explain *all* of the actual findings. He has probably done more complete work in eight years of investigation than any other author. His friends in Russia furnished him with seventy-eight embryos ranging from two millimetres in size to forty-nine millimetres in size. These embryos he cut in serial sections and in the period of five years of investigative work he made wax model reconstructions of each one of these seventy-eight embryos. His findings he showed, with the models, at the sixth and seventh Russian Surgical Congress, and published a monograph in Russian which described his work in detail. Besides the work on embryos he performed 144 autopsies upon the neck region of infant cadavers, in each instance making serial sections of this material, and added serial section studies of fifty-nine adult autopsies. When a man develops a theory upon the basis of such complete and intensive work we must seriously look into and consider his findings. Wenglowski studied the branchial apparatus and the development of the neck and such organs as the thymus and the thyroid glands from these embryos.

THE DEVELOPMENT OF THE BRANCHIAL APPARATUS

In 1825, Rathke studied the branchial apparatus in the embryo of the pig. In 1827, Von Baer found four branchial clefts in the human embryo. In 1877, Cusset stated that the first arch developed on the fifteenth day and the fourth in the first half of the second month. All arches he found tended to join with those of the opposite side. The arch was composed of mesoderm, the outer side covered with flat, and the inner with cylindrical epithelium. All of the arches develop in the region of the base of the skull, but some of them by their rapid growth are forced down into the neck region. The edges of the fourth arches are rounded. They grow together much faster outside than inside. Therefore the epithelium on the outer clefts must disappear faster. If it does not, all sorts of pathological conditions can develop. Before the end of the second month all clefts are closed. The arches meet in the mid-line and form a bridge from side to side. The closure of the outer end of the branchial arches lies in a line drawn from the lesser cornu of the hyoid bone to the sternoclavicular joint.

In 1881, His began his publications. He was the first to work with human embryos, and to make reconstruction wax models. His main advance was in the description of the exact position of the branchial arches. The first arch meets in the mid-line and the lower arches are separated further and further in a young embryo, thereby forming a triangle which he called the "mesobranchial field." As growth takes place the arches telescope over one another, so that the second overlies the third and the third the fourth as seen from without, while from within the pharynx the fourth overlies the third and the third the second. At the same time the third and fourth arches are covered by the wall of the neck so that a depression is caused which His called the "sinus præcervicalis."

This is a projection which develops in the human embryo between the second branchial arch and the primary chest wall. At the beginning of the second month the growth of the branchial apparatus stops. At this time the second arch covers over the sinus. The entrance into the sinus is triangular with the base below and the point above. It is bounded in front or ventrally by the curved edge of the second arch, below by the chest wall, and behind or posteriorly by the lateral wall of the neck. In the eleven- to twelve-millimetre embryo the sinus præcervicalis is completely covered over and has disappeared.

In 1883, G. Born did magnificent and perfect detailed work with a pig's embryo. He showed in his reconstruction models that the anterior part of the tongue develops from the first arch. Opposing His's findings he showed that there was a blind, short prolongation to the third branchial pouch covered by several layers of pavement epithelium. This prolongation he considered to be the thymus anlage.

In 1888, Piersol showed in his work on rabbit embryos that the perforation of the dividing membrane between the cleft and the pouch can occur only in the second branchial cleft.

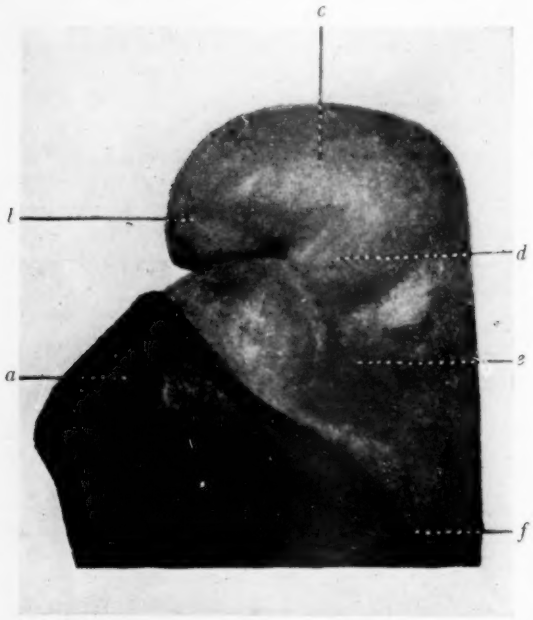


FIG. 1.—2.6-millimetre embryo. Model of upper portion. (Wenglowski.) *a*—Heart region. *b*—Nasofrontal process. *c*—Cephalic end. *d*—Bulge of first branchial arch. *e*—Bulge of second branchial arch. *f*—Cut end of amnion.

In 1889, Von Liessner stated that one had to accept the theory of the patency of the branchial clefts in all types of anomalies. In 1902, Hammar (Upsala) published a work based on the human embryo, but he had used only those of three, five and eight millimetres. Then, in 1912, Wenglowski published the monograph in which he gave the findings of the detailed work on human embryos, beginning with two millimetres and ending with forty-nine millimetres, undoubtedly the most thorough work that has been done on this subject.

Wenglowski began with an embryo of 2.6 millimetres and ended with the embryos in which there was no evidence of the branchial apparatus.

TWO-AND-SIX-TENTHS-MILLIMETRE EMBRYO.—The 2.6-millimetre embryo shows only the first and second branchial arches with an indication of the first cleft. The first arch has distinct free ends anteriorly bordering the mouth opening. The second arch lies close to the anlage of the heart. Between these two arches lies the first branchial cleft. (Fig. 1.) Sagittal section shows even as early as this that the branchial pouches are evident and run in the same direction as the outer branchial clefts. (Fig. 2.)

SIX-AND-FIVE-TENTHS-MILLIMETRE EMBRYO.—Here the relations are much more complicated due to the almost complete development of the branchial apparatus which lies obliquely, from below and anterior, upwards and posterior and almost on top of the upper surface of the heart. In Fig. 3 all four arches and clefts are to be seen.

The first arch.—This consists of two portions. From the lateral portion of this arch the upper jaw develops, and from the anterior portion the lower jaw develops. (Fig. 4.)

The second arch.—This also consists of two portions. The thickened part of the

lateral portion forms the so-called operculum (outgrowth) and covers the third arch. The anterior portion grows to meet the one from the opposite side, and, as shown in the figure, forms a narrow, thin strip.

The third arch.—This is much smaller than the first and second arches, and lies closer to the mid-line than the first and second. More than one-half of it lies buried under the operculum of the second arch. Where the two portions meet, a small operculum also seems to be formed. The medial end gets thinner and thinner and finally disappears completely near the mid-line.

The fourth arch.—This arch is even smaller than the third and lies almost entirely buried beneath the third arch, deep in the recess between the head and the heart region of the embryo. There is an indication here also of two portions. The anterior ends join with the anterior ends of the third arch.

Viewed from in front the first branchial arches meet to form a broad union of the lower jaw. The second arches meet to form a narrow union, and the third arches do not meet in the mid-line but pass upwards to join onto the second arches.

The first branchial cleft.—At the posterior end of the floor of the first cleft is a depression, cylinder-like in shape, ending in a thin membrane. The latter will go to form the external auditory canal. From here the cleft becomes shallower as it goes anteriorly.

The second branchial cleft.—This is almost entirely buried under the operculum of the second arch. The floor is divided into two portions, a lateral and a medial. The lateral portion becomes deeper and narrower, and is separated from the second branchial pouch by a thin membrane. The medial portion becomes very shallow near the mid-line.

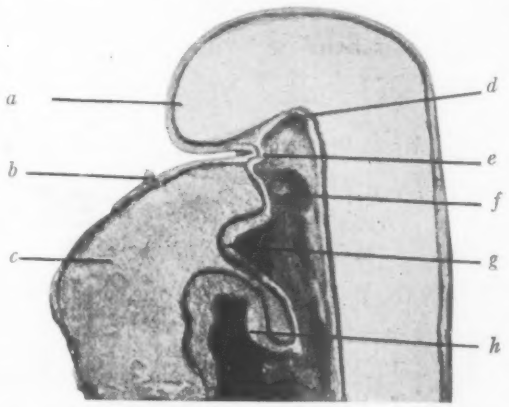


FIG. 2.—Sagittal section of same embryo as seen from within. One sees the upper end of the digestive tract, pharynx and anlage of the respiratory system and liver. (Wenglowski.) *a*—Nasofrontal process. *b*—Cut edge of amnion. *c*—Heart bulge. *d*—Anlage of hypophysis. *e*—Pharyngeal lining. *f*—Second pharyngeal arch. *g*—Anlage of trachea. *h*—Anlage of liver.

The third branchial cleft.—This is similar to the second but smaller and shorter. It also has two portions and is separated from the third pouch the same as the second cleft.

The fourth branchial cleft.—This is even more shallow but wider than the third cleft. It is bent at an angle of 60° and opens anteriorly and laterally. The floor of this cleft is fairly wide. As the fourth branchial arch, so the fourth branchial cleft is hidden in the depression which is formed by the lower border of the third arch and the free arch-like margin of the lateral border of the neck which grows outward and forms an angle at the transition of the head into the chest. This groove was called the sinus præcervicalis by His and the sinus cervicalis by Rabl. The floor of this groove is formed by the fourth branchial cleft. In the embryo studied by Wenglowski the sinus cervicalis is formed by the third arch and the lateral wall of the neck. His and Rabl claimed that the second branchial arch formed part of the sinus, which would make it triangular with the third and fourth arches within it. Wenglowski claims that the second arch plays no part and that only the third branchial arch comes into consideration.

When viewing the branchial apparatus from within the pharynx, one sees the pharyngeal pouches and arches. (Fig. 5.)

The first pharyngeal arch.—This is massive and consists of three parts. They are two lateral portions and a medial portion. The lateral portions are semicircular. The medial arch is separated from the lateral elevations by two depressions. These elevations

narrow towards the front, and from the medial portion of the arch the body of the tongue is later formed. This pharyngeal arch corresponds with the branchial arch and runs parallel with it.

The second pharyngeal arch.—This runs into the posterior pharyngeal wall, and in the mid-line is narrower and does not meet the opposite arch, as the so-called furcula lies between the ends. The direction of the pharyngeal arches is opposite to that of the outer branchial arches. The medial portions of the branchial arches are turned upwards, the pharyngeal arches downward. Thus they cross in the form of a letter "X." The second branchial arch is thicker laterally and thinner medially, while the pharyngeal arch is thinner laterally and thicker medially.

The third pharyngeal arch.—This arch is shorter than the second. Its direction is horizontal and upwards, forming an "X" with the second pharyngeal arch. In the middle of the "X" is the furcula.

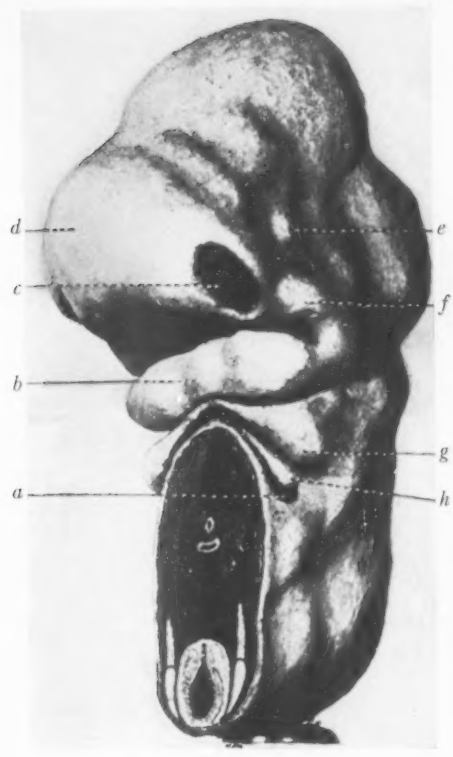
FIG. 3.—Lateral view of complete model of 6.5-millimetre embryo. (Wenglowski.) *a*—Lower extremity. *b*—Tail. *c*—Divided umbilical cord. *d*—Nasal depression. *e*—Eye. *f*—Mandibular process of first branchial arch. *g*—Second branchial arch. *h*—Third branchial arch and sinus cervicalis. *i*—Cardiac bulge. *k*—Upper extremity.

The fourth pharyngeal arch.—This arch is shorter than the third, and wider than the fourth branchial arch. Its direction corresponds to the branchial arch, and its medial end goes into the furcula, where the same divides into two parts to form the entrance into the glottis.

The pharyngeal pouches are better developed but more complicated than the corresponding branchial clefts.

The first pharyngeal pouch.—The lateral portion later goes to form the middle ear and the ear lobe. At one part the pouch and cleft are separated only by a thin membrane. This is near the anterior portion. The outer and medial portion separates the lateral portion of the tongue from the medial pharyngeal arch.

The second pharyngeal pouch.—This pouch is separated from the second branchial cleft near the median portion, but they are close together at the lateral portion, separated only by a thin membrane. This pouch is deeper than the first pouch.



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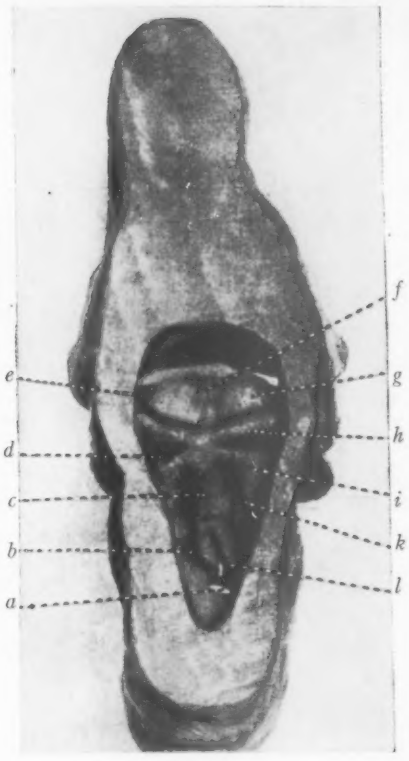

FIG. 5.

FIG. 4.—Head end of same embryo of 6.5 millimetres. (Wenglowski.) *a*—Sinus præcervicalis. *b*—Mandibular process of first branchial arch. *c*—Nasal depression. *d*—Nasofrontal process. *e*—Eye. *f*—Superior maxillary process of first branchial arch. *g*—Second branchial arch with operculum. *h*—Third branchial arch with operculum.

FIG. 5.—Model of same embryo. Posterior portion of head and pharynx have been removed so that anterior pharyngeal wall is visible. (Wenglowski.) *a*—Œsophageal entrance. *b*—Fourth pharyngeal pouch. *c*—Third pharyngeal pouch. *d*—Second pharyngeal pouch. *e*—First pharyngeal pouch with anlage of tongue. *f*—Medial process of tongue. *g*—Lateral anlage of tongue. *h*—Second pharyngeal arch. *i*—Third pharyngeal arch. *k*—Third pharyngeal pouch. *l*—Entrance into glottis.

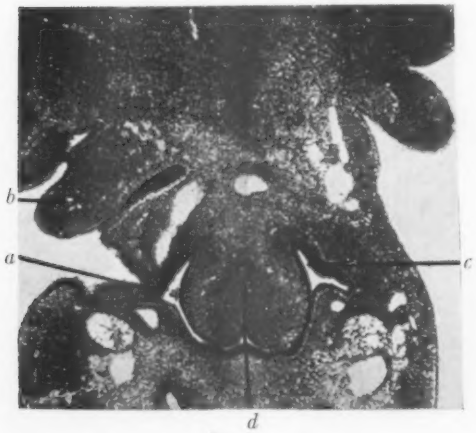

FIG. 6.

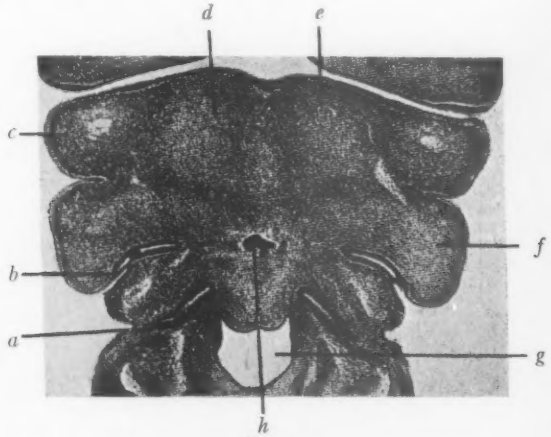

FIG. 7.

FIG. 6.—Microphotograph of cross-section of same embryo above level of glottis. (Wenglowski.) *a*—Grove of præcervical sinus and point of contact of epithelium of sinus and anlage of thymus. *b*—Second branchial arch. *c*—Anlage of thymus. *d*—Epiglottis.

FIG. 7.—Microphotograph of cross-section through tongue and mid-thyroid lobe anlage. (Wenglowski.) *a*—Closing membrane of third branchial cleft. *b*—Closing membrane of second branchial cleft. *c*—First branchial arch. *d*—Right half of anlage of body of tongue. *e*—Left half of anlage of body of tongue. *f*—Second branchial arch. *g*—Pharyngeal cavity. *h*—Anlage of mid-thyroid lobe.

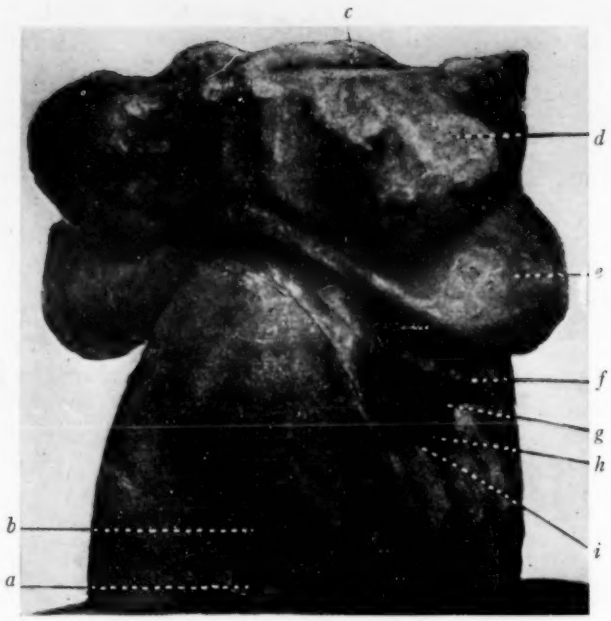

FIG. 8.

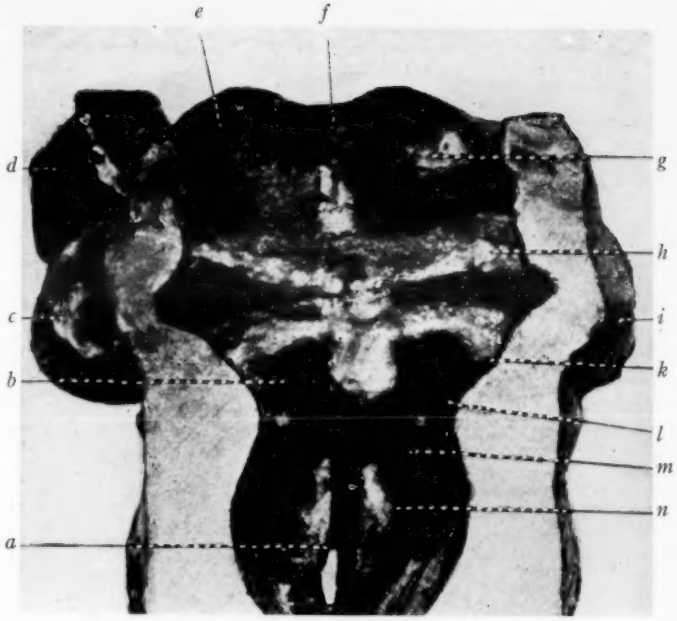

FIG. 9.

FIG. 8.—Model of branchial apparatus of 8-millimetre embryo. The superior maxillary process of first arch and nasofrontal process have been removed. Some of chest wall has been removed. (Wenglowski.) *a*—Œsophagus. *b*—Trachea. *c*—Tip of tongue. *d*—First branchial arch. *e*—Second branchial arch. *f*—Third branchial arch. *g*—Fourth branchial arch. *h*—Fifth branchial arch. *i*—Lateral border of neck.

FIG. 9.—Same model as seen from within. (Wenglowski.) *a*—Entrance into glottis. *b*—Third pharyngeal pouch. *c*—Lateral portion of second branchial arch. *d*—Lateral portion of mandibular process first branchial arch. *e*—Lateral anlage of tongue. *f*—Medial anlage of tongue. *g*—Lateral anlage of tongue. *h*—Second pharyngeal arch. *i*—Lateral process of second branchial arch. *k*—Third pharyngeal arch. *l*—Third pharyngeal pouch. *m*—Fourth pharyngeal arch. *n*—Fourth pharyngeal pouch.

The third pharyngeal pouch.—This is twice as short as the second. It lies deeper and the lumen has an oval form.

The fourth pharyngeal pouch.—This is longer and broader. The dividing membrane between the branchial cleft and the pharyngeal pouch is much thicker.

Microscopically, the branchial arches and the clefts are covered by squamous epithelium. The pharyngeal arches and pouches are covered by several layers of epithelium, in areas composed of ciliated epithelium. The second pouch is rarely ciliated, while in the third the cilia are predominant. In the third pharyngeal pouch where the thymus anlage is, the epithelium is squamous and distinctly hornified. (Fig. 6.) In areas there is simple epithelium, and in other areas there is ciliated epithelium. (Fig. 7.)

Eight-millimetre Embryo.—Here the arches have developed into bigger and more massive structures and thereby the clefts are much narrower. On the left side there are six arches, while on the right there are only five. The second arch lies close to the third, and the third lies close to the chest wall, thereby narrowing the entrance into the sinus of the neck.

The first branchial arch.—This consists of two parts, a medial and a lateral. From Fig. 8 it is seen that it is quite complicated and one can see elevations and depressions, the anlages of the lips and jaws.

The second branchial arch.—This is more massive, especially in its lateral portion. From here it narrows and goes along the lower border of the first arch towards the mid-line where it goes over into the narrow portion and joins with the opposite second arch. At the angle of the two portions is an outgrowth called the operculum, which is, however, smaller than it was in the 6.5-millimetre embryo.

The third branchial arch.—This arch is thicker and more massive. The lateral portion is short. The longer medial portion narrows gradually, and as it does not reach the mid-line it goes over into the lateral wall of the neck. The lower border of the third arch is pointed and forms the upper border of the entrance into the sinus of the neck.

The fourth branchial arch.—This is covered by the third arch and almost invisible from the outside. It runs parallel to the third arch and is one-half its length. The medial end goes over into the lateral wall of the neck.

The fifth branchial arch.—The fifth arch is better developed on the left side than on the right. It is in the shape of a three-cornered prism, where the base is on the lower wall of the sinus and the point is directed upwards.

The sixth branchial arch.—This arch is present in the embryo only on the left side. It is situated at the transition of the lower and anterior walls of the sinus.

In the region of the first to the fourth branchial arches the branchial apparatus is converging. From the fourth to the sixth arches it is diverging.

The branchial clefts on the whole are similar, but the entrance into the cleft is crowded downwards on account of the growth of the lateral portions of the arches.

The first branchial cleft.—In the posterior portion of this cleft is the external auditory canal which in the meantime has developed and is actually separated from the cleft by a small process.

The second branchial cleft.—This cleft has a deep pocket on the left side posteriorly and is separated from the pharyngeal pouch by an oval membrane. On the right side the cleft is equally deep throughout.

The third branchial cleft.—This cleft is longer on the right side than on the left. It occupies the rest of the sinus. On the right side is a deep pocket which ends with an oval small membrane. On the left side it is more shallow and also ends in a membrane with an interposed thicker mesenchyme layer.

The fourth branchial cleft.—This cleft is shallow and is separated from the pharyngeal pouch by a thick mesenchyme layer.

The sinus cervicalis is deeper and larger. The entrance is narrower, not triangular, but elongated and oval. The upper border of the entrance is formed by the lower border

of the third branchial arch. The anterior portion and lower margin are formed by the lateral regions of the neck and the anterior portion of the chest wall. The cavity of the sinus is taken up by the fourth, fifth and sixth arches and clefts. The deepest part corresponds to the upper median pocket of the third branchial cleft. The next deepest portion is the floor of the fourth cleft. The posterior wall is composed of the posterior end of the fourth arch and cleft. The upper wall is the lower surface of the third arch and the floor of the third cleft. The medial wall is mainly the base of the fourth arch, the floor of the fourth cleft and the medial end of the fifth arch. The anterior wall is formed by the lateral wall of the neck, and the lower wall, or the floor of the sinus, is formed by the fifth and sixth arches and clefts.

Seen from within it is much the same as in the 6.5-millimetre embryo. (Fig. 9.)

The first pharyngeal arch.—The anlage of the tongue is much more distinct and is

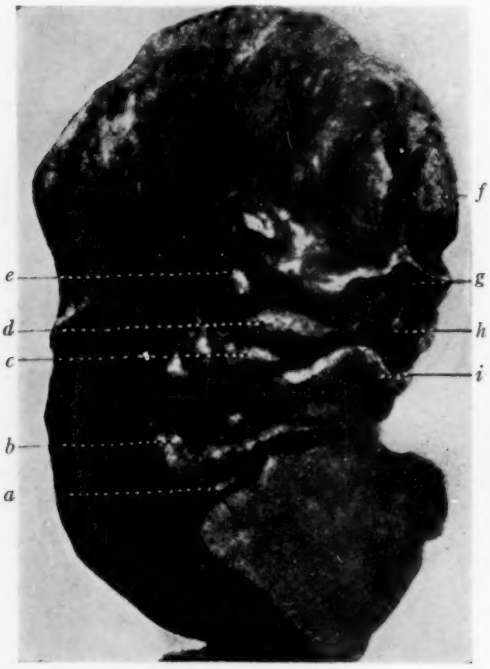

FIG. 10.

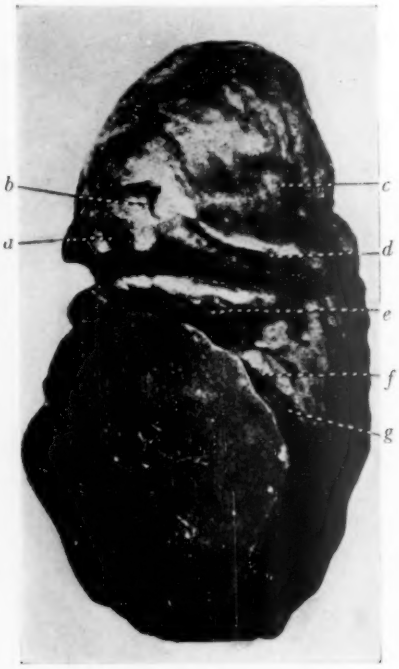

FIG. 11.

FIG. 10.—Model of head of 11-millimetre embryo. Anterior view. Anterior chest wall removed. (Wenglowski.) *a*—Third branchial arch. *b*—Second branchial arch. *c*—Lateral portion of upper lip. *d*—Superior maxillary process. *e*—Eye. *f*—Frontal process. *g*—Nose. *h*—Anlage median portion of upper lip. *i*—Mandibular process.

FIG. 11.—Model anterior head portion 13–14-millimetre embryo. Chest and posterior head portion removed. (Wenglowski.) *a*—Anlage mid-portion upper lip. *b*—Nose. *c*—Eye. *d*—Lateral portion upper lip. *e*—Mandibular process first branchial arch. *f*—Second branchial arch. *g*—Angular curve of neck.

composed of two portions. A lengthened medial process is seen in the medial portion of the tongue.

The second pharyngeal arch.—This arch is much thicker and shorter, but otherwise the same as before.

The third pharyngeal arch.—This is conical and the lateral end is wider and bent downwards. The medial end joins with the rounded body, namely, the furcula of His. It is more massive than the corresponding arch.

The fourth pharyngeal arch.—This is short and thick and corresponds to the branchial arch, but is heavier and thicker. Mesially it ends in the furcula.

The fifth and sixth pharyngeal arches are not to be seen.

The first pharyngeal pouch.—This is a small flat depression between the anterior anlage of the tongue and the second pharyngeal arch.

The second pharyngeal pouch.—This is narrower. Its median portion becomes wider and deeper. Where the pouch meets the pharyngeal wall it turns downward at right angles and ends in a thin closing membrane in the depth.

The third pharyngeal pouch.—This is oval at its opening but gets wider as it gets deeper, so that it is much wider at the bottom than at the entrance. In the lateral portion is a closing membrane. In the posterior lower wall is a process running downward and forward—the thymus anlage.

The fourth pharyngeal pouch.—This has an oval entrance with a wide base. The mesial portion of the floor is taken up by a deep canal, passing forward—the anlage of the lateral thyroid lobes.

The furcula is shorter and more massive.

Microscopically, there are islands of ciliated epithelium in the pouches, especially in the thymus and thyroid canals.

Eleven-millimetre Embryo.—Here the branchial apparatus is in the process of retrogression. The sinus is closed from without so that only three arches are visible. (Fig. 10.)

The first arch is markedly changed and development of the lower lip anlage and lower jaw anlage is marked.

The second arch is quite large, and still conical in shape. Its lateral portion is thicker than the medial portion, and the medial portion joins with the opposite side in a narrow strand.

The third arch is not big. It is parallel to the second arch. Its anterior end narrows and passes and disappears into the antero-lateral wall of the neck. The lower border of the third arch has joined the upper lateral wall of the neck throughout its entire length and displaces the entrance into the sinus.

The branchial clefts are flat and shallow. In the middle of the right third cleft is a narrow passage entering a wide cavity, the remains of the sinus cervicalis. On the left side this canal is not to be found.

Seen from within one notes that the retrogression of the apparatus has progressed markedly. The pharyngeal arches melt one into the other and the pharyngeal pouches are shallower. The tongue takes on a real form, although the body and the base of the tongue are still separate. The base of the tongue consists of two processes joining in the mid-line. These processes are the medial ends of the second and third pharyngeal arches. The lateral ends of these arches pass on to the lateral pharyngeal walls and form the anlage of the alveolar processes. The epiglottis is wide and not well shaped. The anlage of the arytenoid cartilages is noticeable. In the posterior portion of the third pharyngeal pouch are small canals—the thymus canals.

Twelve- to Thirteen-millimetre Embryo.—Further retrogression is noticeable. (Fig. 11.) Only the first and second arches are to be seen. A small depression marks the point of the now absent sinus. The first arch is well differentiated; the second still conical and now horizontal. Its medial ends have joined with the wall of the neck. Due to the displacement of the heart downward the contours of the neck slowly appear.

Seen from within, marked changes have occurred. The pharyngeal arches are low, their contours confluent, and the pouches small shallow grooves. (Fig. 12.) The second and third arches are still to be made out (differentiated), but laterally form the alveolar arches and mesially go to form the tongue. Microscopically, a small cyst is to be seen, the rest of the second cleft, within the mesenchyme, lined with several layers of epithelium. In the sections through the sinus cervicalis region an oval hollow space is seen on the right side lined with squamous epithelium, connecting with the outer world through a narrow canal. On the left side only a small cyst lined with epithelium and surrounded by mesenchyme is to be seen.

FOURTEEN-MILLIMETRE EMBRYO.—Here there is no evidence of the branchial apparatus. Two small, lengthened processes below the lower jaw take the place of the second branchial arch. The neck is more developed.

Seen from within, the tongue is already well formed, but two transverse folds are to be seen near the base, the remains of the second and third arches. (Fig. 13.) The epiglottis and arytenoids are better developed. Two small, rounded canal openings are visible where the third pharyngeal arch passes over on to the lateral pharyngeal wall posteriorly. These are the thymus anlages.

NINETEEN-MILLIMETRE EMBRYO.—No remains of the second arch are to be seen. The neck is better developed and longer. The contours of the neck muscles are to be made out. From within, the well-formed tongue and alveolar arches are to be seen. The epiglottis is separated from the tongue by a depression. (Fig. 14.)

FIG. 12.

FIG. 13.

FIG. 12.—Same model seen from within. Anterior pharyngeal wall. (Wenglowski.) *a*—Œsophageal entrance. *b*—Epiglottis. *c*—Foramen cæcum. *d*—Alveolar process. *e*—Tongue. *f*—Second pharyngeal arch. *g*—Third pharyngeal arch. *h*—Arytenoid cartilage.

FIG. 13.—Model of head end of 14-millimetre embryo. Posterior view. Anterior pharyngeal wall visible. (Wenglowski.) *a*—Entrance into glottis. *b*—Foramen cæcum. *c*—Nasal septum. *d*—Choanæ. *e*—Tongue. *f*—Epiglottis.

Comment.—From the above findings it is seen that the beginning of the development of the branchial apparatus in the human embryo takes place in the second half of the first month. In the course of the second month already, sometimes even in the first half, the branchial apparatus completely disappears. Therefore, its existence in the human being is short, and it does not last more than one month. In the early stages as the heart descends the medial ends of the first arch are near each other. These form the lower border of the primary mouth cavity. The second arches are separated, the third more, and the fourth even more. This forms a triangle with the apex at the mid-portion of the first arch. This triangle is occupied by the heart. From within, an area is formed free of arches, called the mesobranchial field. In this area an elongated, oval, fairly large, bent body is found, with its convexity posterior. This His called the furcula. From it the medial portion of the base of the tongue and the epiglottis develop, and from its lower

portion the glottis itself. During the further growth the arches grow and tend to meet in the mid-line. According to His the branchial clefts and pharyngeal pouches coincide in their direction throughout their length and are separated by a thin membrane. This Wenglowski considers an error. In sagittal sections by His, as found in nearly all text-books, it is shown that the convexity of the branchial arch coincides with that of the pharyngeal arch, and the concavity of the branchial cleft with that of the pharyngeal pouch. In reality, Wenglowski claims this does not occur in the human being, and is only faintly indicated in the three-millimetre embryo where the

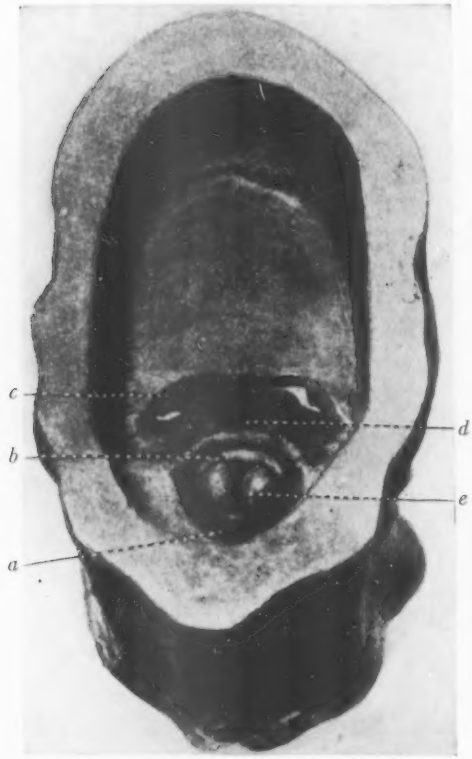

FIG. 14.

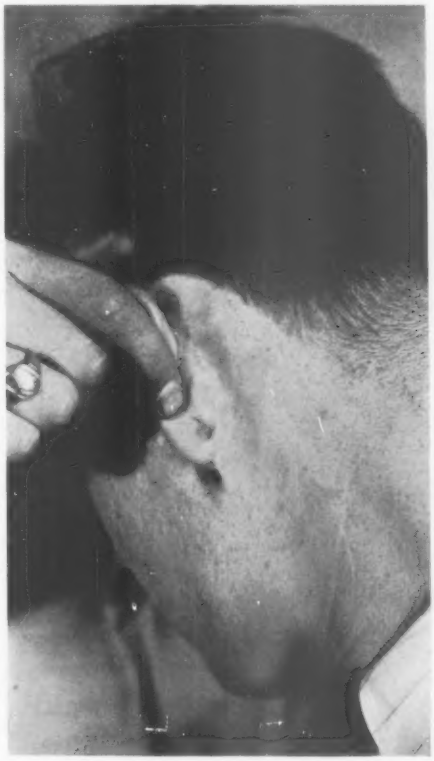

FIG. 15.

FIG. 14.—Model head end of 20-millimetre embryo. Seen from within. (Wenglowski.) *a*—Œsophageal entrance. *b*—Epiglottis. *c*—Mouth cavity. *d*—Foramen cæcum. *e*—Arytenoid cartilage.

FIG. 15.—Author's case of bilateral branchio-genetic cysts developing into fistulæ in the ear lobes and pre-auricular region of the first branchial cleft. The left side had been operated upon before patient came under author's care. Careful dissection and total removal cured the condition. During convalescence the right side suddenly enlarged and had to be operated upon.

apparatus is exceedingly simple and early in development. In the later stages each branchial arch and cleft crosses the pharyngeal arch and pouch. This is caused by the fact that the mesial end of the branchial arches and clefts run forward and upward and orally, while the pharyngeal arches and pouches run posterior and downward and aborally. The bottoms of the clefts and pouches run in different directions as described above in detail, and therefore are in contact only for very small areas where small occluding

membranes are present. The contact point between the clefts and pouches of all the four clefts occurs in the lateral portions. Therefore, the occluding membranes occur in the lateral portions of the clefts. The largest occluding membrane is present in the second cleft.

Wenglowski did not notice a perforation of this membrane in a single well-preserved embryo. The membrane consists of an outer epithelial layer and an inner one with a small amount of interposed mesenchyme. The second and third membranes are thinner, the first thicker, and the fourth still thicker. As the branchial apparatus develops, the membranes get thinner, and are thinnest in the six-millimetre embryo. The retrogression begins in the eight- to nine-millimetre embryo. According to His's theory, the arches disappear due to the thickening of the membranes, which push the clefts from the pouches. This Wenglowski contradicts by showing in what small areas only the membrane is present between the cleft and the pouch. The disappearance of the clefts is caused by two factors: first, by the ingrowth of mesenchyme; and second, by the growth of the arches themselves. The arches grow closer and closer together and thereby narrow the clefts more and more until they finally disappear. This disappearance may be due, first, to the displacement of the epithelium outward by mesenchymal growth, and then again may be due to the adhesion and obliteration of epithelial surfaces in contact. This is best seen in the disappearance of the sinus cervicalis. That the clefts disappear by adhesion and obliteration may be seen from the fact that microscopically one sees epithelial rests surrounded by mesenchyme in the region of the clefts.

It is accepted nowadays that the following structures develop from the following clefts and arches: First cleft—external auditory canal and ear lobe; Second cleft—tonsillar fossa; Third cleft—thymus; Fourth cleft—lateral lobes of the thyroid. First arch—lateral portion of the upper lip, upper jaw, lower lip and lower jaw, and body of the tongue; Second arch—body of the hyoid bone, stylohyoid ligament and muscle, anterior portion of the base of the tongue, arcus palatoglossus; Third arch—greater cornu of the hyoid bone, posterior portion of the base of the tongue, arcus palatopharyngeus; Fourth, Fifth, and Sixth arches—development of the soft parts in the region of the greater cornu of the hyoid bone. (Fig 15.)

The position of the entire branchial apparatus in the earlier stages, as well as the localization of the final rests, point to the fact that it belongs more to the head than to the neck region. In the embryo shown in Fig. 3, the branchial apparatus runs in the vertical position of the body from in front backward and from below upward. In the adult the entire apparatus groups itself, or, better, it rests along the lower jaw and around the hyoid and its cornu. In other words, the direction remains the same as in the embryo. The lower border of the hyoid forms the lower border of all the remains of the branchial apparatus, and nothing below this line has any genetic connection with the branchial apparatus. In spite of the fact that it seems that the branchial apparatus occupies the entire neck in young

embryos, and changes its position as seen in Figs. 3 and 4, in reality the apparatus, with the exception of the first and second arches, does not change its position. The massive nasofrontal process shortens and decreases in size rapidly. The head of the embryo is lifted by the rapid growth and rounding of the head. The lower jaw is lifted somewhat and passes more anteriorly, at which time the heart descends, so that one sees all the facial outlines indicated, the lower jaw appears, and the remains of the second, third and fourth arches run backward and somewhat downward, and not only downward as they appear in Fig. 4. The neck is in its early development. Of course the development of the neck below the hyoid is of importance, as the development above the hyoid occurs at the same time as that of the branchial apparatus.

At first the region above the hyoid is large and that below the hyoid is absent, and right between the second arch and the heart the anterior chest wall will appear. During further development the portion above the hyoid remains almost unchanged. The portion below the hyoid lengthens as the heart descends. The branchial apparatus, however, can influence only the upper neck region, as its position is sharply demarcated by a line drawn through the hyoid bone.

In other words, all congenital anomalies caused by incomplete retrogression of the branchial apparatus *must* be located in the region above the lower border of the hyoid bone.

This means that almost all congenital anomalies of the neck are *not* the result of the branchial apparatus, but come from other factors, even though they may have originated from this apparatus.

THE DEVELOPMENT OF THE THYMUS

In 1831, Arnold found in an eight-weeks embryo two passages on either side of the trachea which he interpreted as belonging to the thymus. Koelliker believed that the thymus developed from the second cleft. In 1881, Stieda stated from experiments on sheep and pig embryos that the thymus developed from the third or the fourth cleft.

His at first believed that it came from the second, third and fourth pharyngeal pouches. He later changed his mind and in 1886 stated that it came from the depth of the sinus cervicalis, and again in 1889 changed his opinion, believing that it came from the branchial cleft.

In 1883, Born showed in a pig embryo that the thymus developed from the posterior portion of the third pharyngeal pouch. Hammar found no evidence of the thymus in a three- to five-millimetre embryo. In the eight-millimetre embryo he found in the corner of the third pharyngeal pouch a pocket running downward and medially, converging with that of the opposite side. He published a picture of an 18.5-millimetre embryo in Kollmann's Atlas, in which he showed the thymus as a long strand running alongside the pharynx and the œsophagus. The upper gland portion was thickened and from the upper end of the thymus a narrow strand passed upward indicating a connection with the pharynx. This is the thymo-pharyngeal duct. The entire thymus looks like an even cylindrical duct passing downward with a thickening at the end, the lower ends close together in a converging fashion.

In 1912, Wenglowski published his findings about the thymus which he studied in serial sections and wax model reconstructions of the human pharynx, trachea, and organs branching from these.

Six-and-five-tenths-millimetre Embryo.—The third branchial pouch is well developed. It is in close contact in its lateral and partial lower wall with the epithelium of the third branchial cleft. In the lateral portion of this pocket on the lower wall a bag-like depression passes downward and somewhat laterally. In the model one sees that from the lowermost portion and from the lateral wall of the fourth pouch two large processes pass forward. These are the anlages of the lateral thyroid lobes. (Fig. 16.)

Somewhat more forward and lateral to these is the third branchial pouch, which laterally passes into the third cleft. From the angle of this pouch a bag-like depression arises somewhat downward and posterior, and appears to cross the thyroid anlage. This is the thymus anlage.

Microscopically, one finds that the epithelium of this pouch is so closely in contact with the epithelium of the cleft that one cannot differentiate it. At the medial end one

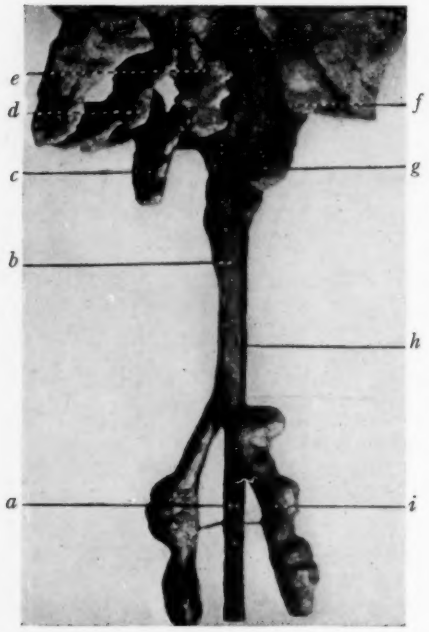

FIG. 16.

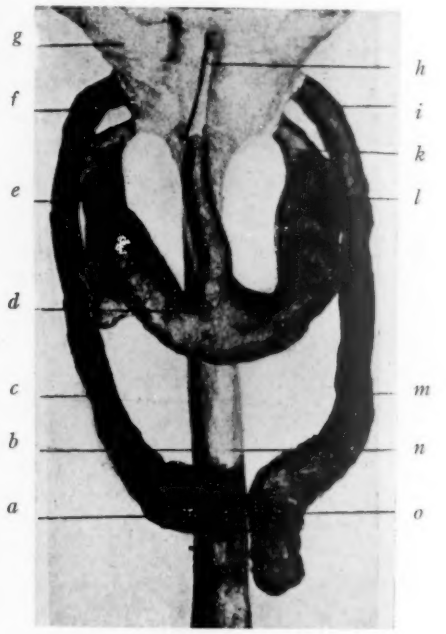

FIG. 17.

FIG. 16.—Model of œsophageal lumen and branchial apparatus of 6.5-millimetre embryo. Model straightened for greater clearness. (Wenglowski.) *a*—Œsophagus. *b*—Trachea. *c*—Lateral thyroid lobes. *d*—Thymus anlage. *e*—Mid-thyroid anlage. *f*—Thymus anlage. *g*—Lateral thyroid anlage. *h*—Œsophagus. *i*—Left lung anlage.

FIG. 17.—Model of pharynx, œsophagus, trachea and organs developing from them in a 14-millimetre embryo. (Wenglowski.) *a*—Thymus. *b*—Œsophagus. *c*—Thymus duct. *d*—Mid-thyroid lobe. *e*—Lateral thyroid lobe. *f*—Thymus duct. *g*—Pharynx. *h*—Thyro-glossal tract. *i*—Thymus duct. *k*—Lateral thyroid lobe duct. *l*—Lateral thyroid lobe. *m*—Thymus lobe. *n*—Trachea. *o*—Thymus.

notices a thickening of the epithelium from which a pouch passes downward into the mesenchyme. On the lateral wall are several layers of flat epithelium, while on the medial wall is ciliated epithelium. (See Fig. 6.)

Eight-millimetre Embryo.—Here the thymus anlage is more developed. It passes downward and posteriorly, crossing the thyroid anlage. Microscopically, the hollow thymus anlage connects only with the pharynx through the mesial end of the third pharyngeal pouch. A narrow canal goes from the wall of the pharynx, in the region of the third pouch, laterally, and is in close contact with the epithelium of the sinus. It then bends at right angles and passes medially and posteriorly. In this manner the thymus anlage communicates with the pharynx through a very complicated angulated

canal whose angle closely touches the epithelium of the sinus. This angulated form is important and this type is seen and explains certain types of pathological findings. The posterior lateral wall of the thymus anlage is thicker than in the previous embryo, and grows into the mesenchyme in irregular fashion. The anlage lumen is lined with stratified squamous epithelium, but ciliated epithelium is also found, especially above the angulation.

Twelve- to Thirteen-millimetre Embryo.—The thymus is well developed. It is a long strand with two limbs, thickened at its lower end. The upper short limb passes downward in the space between the lateral ends of the third and fourth arches. It lies closer to the third arch, and at the point where this curves onto the lateral pharyngeal wall it forms a fold, later the arcus palatopharyngeus. From here the upper limb passes almost horizontally outward and downward and comes very close to the outer skin of the neck. Here on both sides the thymus anlage lies close to the remaining little cysts, remainders of the sinus cervicalis of the neck. These little cysts are behind and lateral to the thymus anlage. Here the thymus anlage becomes angulated and at a right angle passes into the lower limb, which runs downward, forward and medially. The lower end is thickened irregularly. The entrance of the anlage into the pharynx is slightly anterior to the anlage of the lateral lobes of the thyroid. From here it crosses the thyroid and passes onto the outer posterior surface of the same and follows it. The thickened ends pass down below the thyroid anlage.

The thymus anlage has a lumen throughout, situated near the anterior wall of the anlage. Only at the lower thickened end is this lumen absent. The lumen is semilunar in shape with its convexity postero-lateral and lined with flat epithelium. Here and there are islands of ciliated epithelium. At the lowest end the epithelium changes into round-cell masses. The thymus canal enters the lateral pharyngeal wall in the third pharyngeal arch where the latter curves upward over the base of the tongue.

Fourteen-millimetre Embryo.—Here the thymus is at its greatest development. All parts are still present. There are two limbs, one the short horizontal portion, and one the long strand, which is vertical. The angle between the two limbs is much less acute and much more rounded. The vertical portion is very long and equally thick, and follows the lateral border of the thyroid lobe. It passes down below the thyroid and the lower ends of the two sides almost join in the mid-line. On the right side is a narrow lumen throughout the extent, while on the left side there is a lumen only in the upper and middle third, without an opening into the pharynx. Small cysts are to be seen microscopically, lined with ciliated epithelium. On the left side no lumen is to be found microscopically between the upper and middle thirds. The canals enter the pharynx at the posterior border of the third pharyngeal arch where this passes from the lateral surface of the tongue onto the lateral wall of the pharynx. The upper third of the lumen is lined with ciliated epithelium. Below this is squamous epithelium with islands of ciliated epithelium. (Fig. 17.)

Sixteen-millimetre Embryo.—Two changes have taken place. The lower end takes on the form of a gland. At the same time retrogression takes place at the upper portion of the anlage. As seen in the model, the strand is divided into several divisions. At the anlage near the pharynx a conical process remains behind at the posterior end of the third pharyngeal pouch. This rest is lined with polyhedral epithelial cells, and at the anterior wall is a narrow lumen which almost reaches the pharynx. Near the pharynx there is squamous epithelium. Further away there is ciliated epithelium. Then there is an interval, and behind the thyroid lobe it again appears and ends in a dilated end. The rest near the pharynx is entirely missing on the right side, and the anlage begins only behind the lateral lobe of the thyroid. The lumen is present only on the right side in certain areas, and on the left side is not completely present any more. Some little

cysts are present lined with ciliated epithelium. The lumen near the anlage is still semilunar in shape. This is caused by an ingrowth of cells into the lumen which presses it against the wall of the canal. The direction is still the same, downward and forward, on one side along the dorso-lateral border of the thyroid, and on the other side along the anterior border of the sternocleidomastoid down to the sternum. (Figs. 18, 19.)

Nineteen- to Twenty-millimetre Embryo.—Retrogression is more advanced. On the reconstruction model the upper and lower ends have a different appearance. The lower end is knobbed, irregular and thickened. The upper end is even, cylindrical and strand-like. The left side is distended from the upper border of the thyroid to the sternum. It is smooth and only in its upper third is it indented. Inside is a flattened canal throughout its length. On the right side the upper half is absent, and the anlage begins at the upper end of the thyroid and runs downward into the gland substance. The cylindrical portion has a canal throughout. Cross-section shows two thymus canals, unlike in size, lined with ciliated epithelium, with a thin dorsal wall and a thickened ventral wall. In other areas stratified flat epithelium is present. The course and direction are typical. The

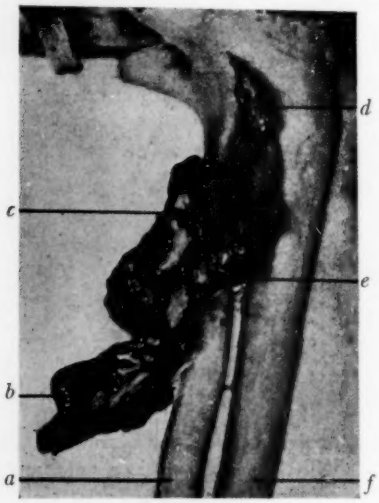

FIG. 18.

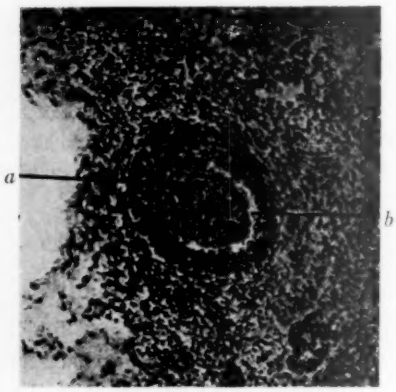

FIG. 19.

FIG. 18.—Lateral view of model of pharynx, œsophagus and organs developing from same in 16-millimetre embryo. (Wenglowski.) *a*—Trachea. *b*—Thymus. *c*—Thyroid. *d*—Rest of upper portion of thymus duct. *e*—Thymus duct. *f*—Œsophagus.
FIG. 19.—Microphotograph of cross-section through the thymus duct in 16-millimetre embryo. (Wenglowski.) *a*—Ingrowth of glandular cells into lumen. *b*—Lumen of thymus duct.

lower end lies in the upper portion of the chest cavity. In the retrogression process a certain orderly fashion occurs. The uppermost portion disappears first, and from there on the retrogression passes downward. It is unusual to find thymus rests near the pharynx in two- to three-months embryos. Dorsal to the lateral lobes of the thyroid and below rests are frequently seen. (Figs. 20, 21.)

These rests have a fairly regular histology. In the centre or near the ends are hollow spaces lined by either squamous or ciliated epithelium, or both. Outside of this lymphoid cells are often found. The nearer the pharynx the more frequently ciliated cells are found. The nearer the sternum the more frequently squamous epithelium is found.

The thymus develops later than the medial and lateral thyroid lobe anlages. It is first seen in a 6.5-millimetre embryo. It is due to the close

proximity of the thymus anlage to the epithelium of the sinus cervicalis that His believed that the thymus originated from the sinus, while Wenglowski shows that it develops from the third pharyngeal pouch.

Piersol, Prennart and Hammar confirmed the close association between the sinus epithelium and the thymus anlage. The thymus canal between the sinus and the lateral wall of the neck is lined with ciliated epithelium. The pharyngeal opening and further down is lined by squamous epithelium. The walls below the angle are irregularly thickened and the cells take on lymphoid characteristics. The canal reaches the level of the sternum. Below this the gland develops without a canal and lies anterior to the great vessels within the chest.

If the thymus anlage did not retrogress it would go from the pharynx laterally and slightly downward to the area between the angle of the jaw and the ear lobe. From here it passes downward and forward and medially, lying close to the lateral border of the thyroid gland and medial to the border of the sternomastoid muscle, down to the sternum where it passes into the actual gland substance.

This complete structure rarely occurs later in life, and in embryos only until the second or third month. The neck portion totally disappears. Sometimes it does not completely disappear and segments may persist during life and not cause any trouble. The lower portion persists more frequently than the upper, and very rarely the portion close to the pharynx. It has also been definitely established that if rests are still present at the third to fourth months of embryonic life, they will persist throughout life in a latent condition.

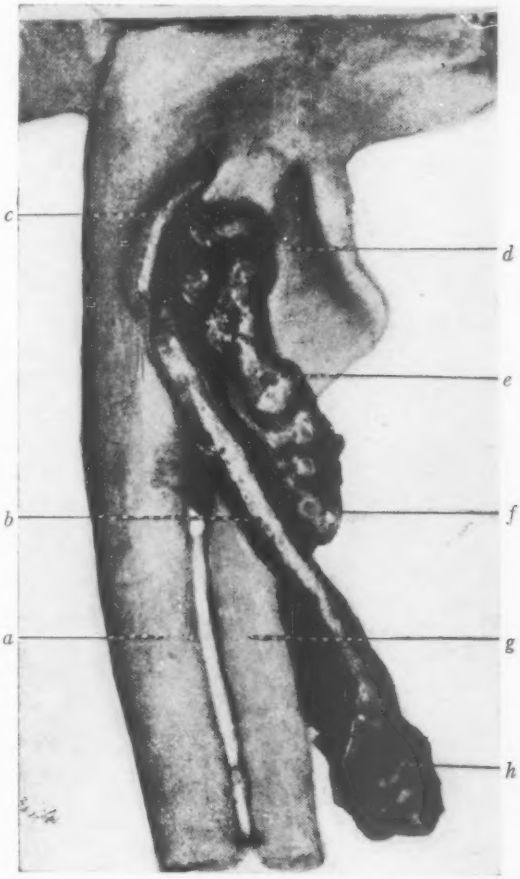

FIG. 20.—Lateral view of model of pharynx, œsophagus, trachea and organs developing from the same in 20-millimetre embryo. (Wenglowski.) *a*—Œsophagus. *b*—Thymus duct. *c*—Rest of upper end of thymus duct. *d*—Glottis. *e*—Lateral thyroid lobe. *f*—Mid-thyroid lobe. *g*—Trachea. *h*—Thymus.

Wenglowski examined the neck of ten adult bodies by careful dissection, with microscopical examinations of all suspicious tissues. He also examined

the bodies of sixty-five infants and did serial sections of the necks. Of the adults two cases were found with thymus anlage rests—some cysts lined with ciliated epithelium and lymphoid tissue in the walls. Of the sixty-five infant bodies rests were found in the serial sections twenty-one times. Twelve times they were found in the lower half of the neck, measuring the half on the sternomastoid muscle, and seven times in the upper half, and twice close to the pharynx.

Histologically, these rests are small canals or cysts, frequently with squamous epithelium, sometimes ciliated and sometimes mixed. The walls are lymphoid in character, similar to the thymus. Hassal's bodies are sometimes found, usually when in the lower half of the neck. In two cases close to the pharynx there were cysts, cylindrical to the point close to the posterior tonsillar fossa, with clear content and mucous glands in the wall, with ciliated epithelium in the lining.

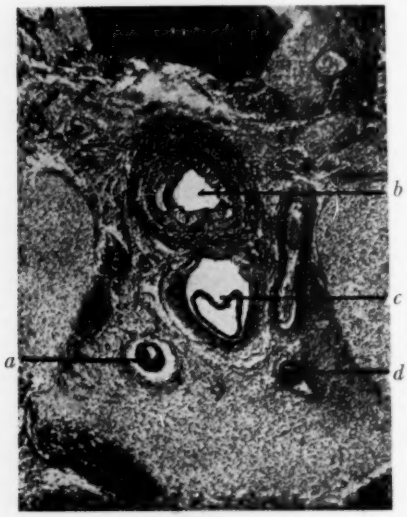

FIG. 21.—Microphotograph of cross-section of neck of 20-millimetre embryo. (Wenglowski.) *a*—Thymus duct. *b*—Œsophagus. *c*—Trachea. *d*—Thymus duct.

Thus we see that a complete canal can persist in the adult and cause pathology early. Also, this canal can be divided into segments. Close to the pharynx mucous glands can be present in the wall, torn off from the epithelium of the pharynx just the same as in the middle thyroid anlage, but they are not carried into the depth as the anlage is in the bottom of the third pouch and not from the lateral wall of the pharynx.

With careful observation Wenglowski found thymus rests at the time of surgical operation upon thyroid conditions and other neck conditions, which microscopically were proven to be thymus tissue.

The Development of the Lateral Thyroid Lobes.—Born demonstrated by means of the reconstruction models that the lateral lobes of the thyroid come from a different anlage than the median lobes. He stated that the anlage of the lateral lobes was to be found in the fourth pharyngeal pouch. Wenglowski found no evidence of an anlage in an embryo of three millimetres. In a 6.5-millimetre embryo there is evidence. The anlage is conical, narrow below and broad at the attachment to the fourth pouch. Its direction is forward and somewhat downward. Microscopically, there is a lumen lined with several layers of epithelium with proliferation of the cells in the wall distal to the pharynx. The lateral lobes grow downward and forward and approach the median lobe.

In an eight-millimetre embryo the anlage is bigger and more prism-like, wider and bigger below and more narrowed above at the pharynx. The lumen is narrowed above and somewhat wider below, but not as large as in the 6.5-millimetre embryo.

In the fourteen-millimetre embryo the attachment to the pharynx is only by a narrow, thin strand. The lobes widen and thicken below and go to meet the median lobe.

Microscopically, on the left side a small canal is seen connecting with the pharynx. On the right side the canal is obliterated and replaced by cells.

In the sixteen-millimetre and nineteen-millimetre embryos the lateral lobes of the thyroid are completely separated from the lateral pharyngeal wall. The primary canals and its rests have completely disappeared. The gland descends downward and becomes a unit with the middle lobe. In older embryos and in autopsy material the canals were never found to persist. However, accessory thyroid tissue was found between the tips of the lateral lobes and the situation of the anlage at the pharynx. Occasionally one finds thyroid tissue in the outer wall of the œsophagus which has been dragged there and imbedded by the rapid growth of the œsophageal musculature.

Thus the lateral lobes of the thyroid develop earlier than the thymus. In the 6.5-millimetre embryo the lateral lobes of the thyroid are well developed while there is no evidence of the thymus. The thyroid lobes develop from the epithelial pocket in the floor of the fourth pharyngeal pouch. The lobes grow downward and inward and become a firm unit with the median lobe. They take on typical thyroid structure histologically, and lose the lumen which is present in the anlage. They completely separate from the pharyngeal wall.

Theoretically the lumen or segments of it might persist, but in 150 autopsies Wenglowski did not find it a single time. Abnormally placed thyroid tissue with pathology is the only condition which can be related to the anlage of the lateral lobes of the thyroid.

Clinical Observations.—Kostaniecki and Milecki accepted the branchial theory. Many cases were described that could not be explained, however, by the branchial theory, and not a single one that in its entirety could be attributed to the second branchial cleft. The relation with the glossopharyngeal nerve was considered as very important, and therefore etiologically it was attributed to the second cleft, but in many cases (Karewski) there were no relations for proximity of the lateral tract with the nerve and the fistula.

Watson, Karewski and Hildebrand, *etc.*, showed that the fistula passed below the stylopharyngeal muscle and usually passed downward at the posterior border of this muscle. This muscle is an important landmark of the third branchial arch. The direction and position of the muscles are as constant as of the nerves and blood-vessels which Kostaniecki and Milecki accepted as the constant findings. They did not take the muscles as a constant finding. In reality the muscles and nerves are more constant than the vessels.

Microscopically, the finding of ciliated epithelium and also squamous epithelium in the wall of a complete or incomplete fistula speaks against the perforation of the closing membrane, with the accepted theory (Kostaniecki and Milecki) that the pharyngeal pouch is lined with ciliated epithelium and the branchial cleft with squamous epithelium.

In other words, it was necessary to juggle anatomical findings in order to explain histological findings in these fistulæ and cysts. The theory of Rabl and his duct cannot explain the fistulæ with openings near the sternum, and His's theory of the second branchial cleft taking part in the sinus cervicalis and the latter then breaking through to the outside must of necessity confine the external opening by the location of the sinus. This is fixed by the third and fourth branchial arches at the dorso-lateral border of the hyoid.

Lower than this the sinus cannot go because it would be prevented by the arch which is fixated at this point.

Kostaniecki and Milecki's theory of the second cleft did state that the inner opening was often in the region of the posterior tonsillar fossa, and it made them think of the possibility of the third pouch as the origin. However, they discarded this theory, as the third pouch is small, and they stated that the lateral fisula had no relation to the thymus. The outer opening, according to Kostaniecki and Milecki, was dependent on the perforation of the closing membrane and the degree of infection. In reality there is a definite and constant finding of the outer opening along the anterior border of the sternomastoid muscle and in the angle of the latter and the sternothyroid and sternohyoid muscles.

The course of the complete fistula is also always a constant one, namely, arched. The arch is always constant below the angle of the jaw about the middle of the posterior belly of the digastric muscle. From here the upper portion goes inward and upward and the lower portion downward and inward along the medial border of the sternomastoid muscle. If one examines the direction of the second branchial cleft and pharyngeal pouch one does not find any likeness to the direction of the course of the fistula.

The relation to the carotid vessels is also of interest. As the external carotid is the axis of the third branchial arch, then the second branchial cleft would have to pass between the external and the internal carotid arteries to open externally. In reality, the fistula does not pass through this space but lies anterior and close to the sheath of these vessels, and from there goes in the direction of the pharynx.

Therefore, to explain the lateral cysts and fistulæ by the branchial theory is so artificial and shows so many erroneous conclusions that it seems we all ought to agree with Wenglowski and once and for all discard the branchial theory as the etiological factor, and stop calling these conditions branchiogenetic cysts and fistulæ. All cysts or fistulæ above the hyoid level might come from the branchial apparatus. Everything below this level must come from other sources. Anatomically, it seems impossible to think of the possible persistence of a cleft beyond the second embryological month. A cyst resulting from it is possible during the first or second embryological month, but not easy to imagine in adult life. The branchial structures obliterate so thoroughly that a complete fistula is not easy to imagine with the breaking through of a cleft to the outside. It is possible to imagine epithelial rests but a complete fistula from a cleft is difficult to understand after the second embryological month. After that, in adult life, clefts cannot exist as the surrounding tissues continuously grow closer and closer together.

The thymopharyngeal duct, however, will explain all the clinical findings. Its course is exactly that of the findings in a patient. Around the arch of the duct are the sinus rests. Above the arch the duct forms a part of the third pharyngeal pouch. Below the arch it forms a part of the actual thymus duct.

A duct with an inner opening soon infects itself and usually breaks through to the outside early, and then a cyst changes into a fistula.

Microscopically, the fistulæ and cysts coincide with the thymus anlage findings. One finds areas with stratified squamous epithelium mixed with ciliated epithelium, and in the walls encysted striated muscle fibres, cartilage and mucous glands. Lymphoid tissue is also present in large amounts which is not found in the mid-line ducts or cysts, but is found in thymus tissue and thymus ducts.

The etiology of mid-line and lateral cysts and fistulæ is different insofar as the mid-line are epithelial rests dragged in by the mid-thyroid anlage, which has no duct, and are not from the anlage itself. The anlage grows rapidly from the floor of the mouth as a solid strand and drags in the surrounding structures, while the thymus anlage grows from the depth of the third pharyngeal pouch outside the pharynx, and does not grow as a solid strand but as a depression which later changes into a canal.

While the mid-thyroid anlage leaves behind it the foramen cæcum, the thymus anlage leaves no mark behind as the third pharyngeal pouch completely obliterates.

One often sees complete lateral fistulæ, but complete median fistulæ do not exist.

It makes no difference where a thymus-duct cyst breaks through to the outside—either near the pharynx or near the sternum along the sterno-mastoid—the course of the thymus duct always remains the same.

Virchow and Koenig had a case which they could not explain by Wenglowski's theory. Wenglowski, however, does explain the cases as being a combination of a thymus duct and an additional abnormal external ear into which the thymus duct perforated due to infection, in addition to the external opening.

Koenig also had a case in which the internal opening was in the glottis. The question is whether this case was due to secondary internal perforation following inflammation and repeated operations, or whether it was primary and due to a persistent anlage duct of the lateral lobe of the thyroid which would enter internally at the entrance to the glottis.

As to the age incidence, complete fistulæ are a condition mostly of youth. Children are often born with it or it develops during the first year of life. Lateral cysts develop later in life.

The external opening is slit-like. It is often difficult to introduce a probe. The internal opening is small, in the tonsillar fossa behind or just in front of the posterior pillar. Heusinger had a case in which the external opening was big enough to admit a finger tip. The lower end of the canal persists more often than the upper end, as retrogression takes place from above downward.

Complete, internal incomplete, and external incomplete fistulæ are found.

Microscopically, one finds squamous epithelial cells and flat epithelial cells, which go over into ciliated epithelium, which also may consist of

several layers. Similar to the findings in the thymus duct epidermoid findings are due to sinus rests which occur at the angulation area where the thymic duct touches the sinus. If a sinus rest does not break into the lumen of the thymus duct, then a cyst or fistula may develop similar to a dermoid, with hair, coarse material, and other attributes. These deep-seated dermoids are not rare in the neck. Lateral cysts and fistulæ are rich in lymphoid tissue. This shows their close relation to the entoderm and to the thymus. The ectodermal cysts are without lymphoid tissue and come from the sinus rests. The contents of the cysts with ciliated epithelium are mucoid; with squamous epithelium there is an admixture of cast-off epithelial cells.

Thus the clinical and anatomical characteristics, as well as the microscopical structure of lateral cysts and fistulæ, not only are closely related to the embryological anatomical examinations, but also confirm them. We must then recognize that the cysts and fistulæ are closely related to the thymus canal. The structure microscopically is altered by the degree of inflammation.

We should, therefore, give up the name of branchio-genetic cysts and fistulæ, and call them lateral cysts and fistulæ.

Treatment.—Surgery is the only dependable cure. If a complete or incomplete tract exists, total excision of the tract is necessary. (Fig. 22.)

Two principles of operation have been described by von Hacker and by Fritz Koenig. In these the tract is inverted into the pharynx. This inversion is possible when the tissues are not rigid or fixed. In unfavorable cases in which there is firm fixation near the pharynx, Koenig has described a method in which he dissects the tract as high up towards the digastric as possible and then by blunt finger dissection towards the pharynx. The probe enters the tract from the neck wound and is pushed towards the mucous membrane anterior to the tonsil and at its lower end. An incision is made into the mucous membrane and the probe is pushed through. The wall of the tract is fastened to the fenestrated end of the probe with a ligature or suture, and it is drawn through the opening into the mouth cavity. In other words, it is led through but not turned inside out, as in the favorable cases. The tract is pulled taut and a suture is placed through the opening and through the mucous membrane. The fistula is then cut away, leaving a short stump which curves around the base of the tonsil. The posterior end is in the tonsillar region and the anterior end is at the newly made opening through the mucous membrane. With this method no important structures are injured or compressed. (Fig. 23.)

In cases in which there have been repeated attacks of suppuration with extensive changes and induration, surgical excision is difficult and attended with considerable risk. It should be done only when there is reasonable assurance that it can be completed successfully. Otherwise, conservative treatment should be instituted for the inflammatory condition.

Complications.—Certain changes can occur in the walls of the cysts either

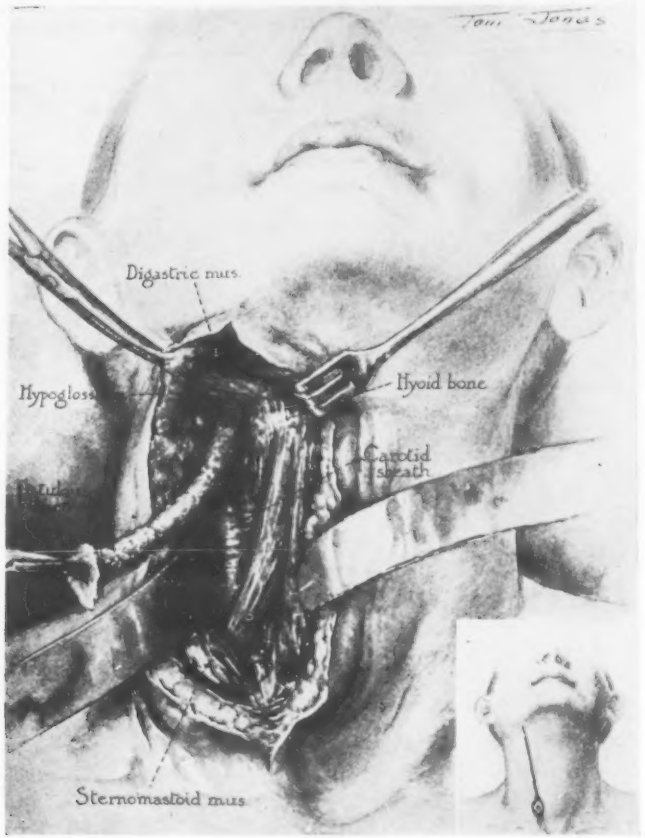

FIG. 22.

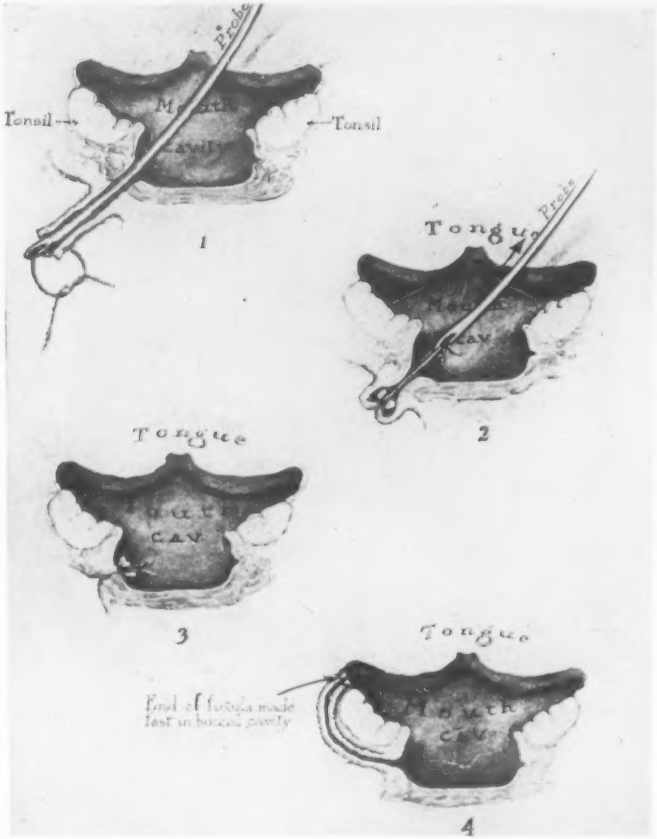

FIG. 23.

FIG. 22.—Drawing of lateral fistula of neck, showing course along the median border of the sternomastoid muscle and arch of duct in mid-portion of the posterior belly of the digastric toward pharynx. (Christopher.)

FIG. 23.—Drawing showing method of treating the inner end of a lateral fistula. 1, 2, 3 show method of von Hacker by inverting the duct with a probe. 4 shows Koenig's method of leading duct around the tonsil. (Christopher.)

real branchio-genetic in origin or true lateral cysts which may be classified as complications:

(1) *Inflammation.*—This may form an abscess of the cyst. (2) *Blood-vessel Changes.*—These may produce blood cysts due to injury. (3) *Cyst Adenoma.*—This may develop from glandular elements coming from the entoderm. (4) *Lymphangioma.*—These arise from lymphatic elements in the cyst wall. (5) *Chondroma.*—This develops from misplaced heterotopic tissue. (6) *Teratoma.*—There is no explanation to explain the presence of epithelial structures such as bone, teeth, muscle, fat, nervous tissue, complete and incomplete small fetuses. These often appear in the mouth or the pharynx, notably in the tissues adjacent to the second pharyngeal pouch. (7) *Carcinoma.*—This has been especially well described in the treatise on surgery of the neck in Nelson's Loose Leaf Surgery, written by Dr. George H. Semken.

Cancers in lateral cysts and fistulæ of the neck are rare, but in the epithelial rests of the sinus of the branchial system they are not uncommon.

Men have it more frequently than women. It develops in adult life, and is of the squamous type of epithelioma. Clinically it is carcinoma. It is a solitary, hard, fixed mass, with rapid growth and early attachment to the great vessels, displacing the neighboring tissues in expansile growth. Later the tumor softens in the centre and eventually breaks through the overlying skin in a fungating mass. Regional lymphoid node metastases are a regular finding. Distant metastases are rare. Pain begins early, due to the close relation to the nerves.

Differential diagnosis must lie between lipoma, which has a different texture and consistency; lymphatic hygroma, which has a different location; tuberculous lymph-node, from which it is difficult to differentiate; and metastatic lymph-node carcinoma, which means a careful search for a primary lesion at the root of the tongue, nares, pharynx, hypopharynx, larynx and œsophagus. As epithelial inclusions at the base of the tongue are frequent, the primary lesion may be beneath the surface, and a careful inspection and palpation must be made. In the lateral region carcinoma of the accessory thyroid or parathyroid glands may occur. Branchiogenetic carcinoma has early fixation to the great vessels and is more lateral than the thyroid or parathyroid carcinoma. Primary lymph-node tumors and Hodgkins' lymphoid granuloma are softer in consistency and less firmly fixed.

The treatment of branchiogenetic carcinoma is careful, complete removal.

(*To be continued*)

MANAGEMENT OF SKULL FRACTURE INVOLVING THE FRONTAL SINUS

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Much has been written concerning the immediate management of frontal sinus fractures. The various complications of the condition have been studied. Dandy (1926)[1] reviews the subject of pneumocephalus and ascribes to fractures of the frontal sinus region an important rôle in the genesis of the condition. Especially if associated with cerebrospinal rhinorrhœa, the possibility of meningeal and cerebral infection and its prevention have been discussed by Peet,[2] Teachenor,[3] Andruss,[4] Naffziger[5] and others. Teachenor, in particular, is very radical in his treatment of fractures of the frontal sinus. He believes in operating on all cases in order to prevent infection. Robb (personal communication) states that in the majority the treatment should be conservative.

In the present paper an analysis of one hundred twenty-five cases of frontal sinus fracture is made, together with a statement of the type of treatment and the results therefrom. This group constitutes a portion of a series of over 2,600 cases of skull fracture seen at the Detroit Receiving Hospital from 1925 to 1929, inclusive. It may be seen, therefore, that fractures involving the frontal sinuses are quite rare, constituting approximately 5 per cent. It is possible that a certain number of this total group may have had fractures in this situation and are not included in this series, because they died within an hour or two after entrance into the hospital, and the sinus fracture probably had nothing to do with their death. Granted the patient's condition permits, he is usually X-rayed within twelve hours after admission. The cases here described have been mostly diagnosed by X-ray. A few had evident compound fracture into the sinus region.

Discharge from the Nose.—In the present series, there were seventy-one cases with no associated bleeding from the nose. Among fifty-two there were evidences of bleeding which ceased in the course of about a day. In two there was continuous oozing of cerebrospinal fluid and blood; two days in the one case and one day in the other. Of the cerebrospinal group, one died with no evidences of meningitis and the other recovered without any operative procedures being resorted to. Cerebrospinal rhinorrhœa is a serious complication in fractures of the anterior fossa and several investigators deem it advisable to interfere in such instances in order to prevent the infection of the meninges. Campbell,[6] among others, has reviewed several cases of spontaneous and post-operative rhinorrhœa where the incidence of meningitis is quite high, but a certain number of these cases did get well with no operative intervention. In fractures of the frontal sinus, rhinorrhœa is probably more

rare than in case of fracture involving the floor of the anterior fossa of the skull.

Incidence of Brain Injury.—A fracture of the frontal sinus region may be completely free from associated signs of brain involvement. As a matter of fact, the majority of our cases have come in conscious and remained so throughout their stay in the hospital. Nineteen gave no history of unconsciousness following their accident and their recovery was uneventful. Fifty cases showed post-traumatic headaches with a short period of unconsciousness following their injury. Among fifty-one there was a period of unconsciousness ranging from an hour to several hours followed by drowsiness lasting several days. Five had serious brain damage as evidenced by the state of unconsciousness and the neurologic condition of the patient. It is noteworthy, however, to observe in this group that more than one-half the cases had very slight brain damage. Teachenor (1926) reports eighteen cases of fractures of the frontal sinus region, practically all of which were moribund, with a mortality exceeding 65 per cent. It is true that the series presented in this paper comprise only those who had been X-rayed and had positive evidence of fracture; hence it is possible that a certain number of the cases who entered the hospital in such a serious condition that raying was inadvisable and who died in the course of twenty-four to forty-eight hours may have had fractures involving this region. The number of such cases was very small.

Location of the Fractures.—Of these one hundred twenty-five cases, the fracture involved the right sinus in fifty-eight instances and the left in forty-nine. There was bilateral involvement among eighteen. There were three cases of compound fracture. A certain number showed definite depression; there were thirteen involving the right sinus; eight, the left; and ten, both sinuses. It is noteworthy to observe that in the majority, the line of fracture extended toward the vertex and hence both the anterior and posterior walls

TABLE I

Analysis of One Hundred Twenty-five Cases of Frontal Sinus Fracture

Discharge from nose			Brain injuries				Distribution of fractures				Depressed fractures			Extension of sinus fracture			Operations		Results		
None	Epistaxis	Cerebrospinal Rhinorrhœa	None	Slight	Definite	Severe	125 cases				31 cases			Into vertex	Into base	Outer wall of sinus only	3		Recovered		
71	52	2	19	50	51	5	Right	Left	Bilateral	Compound	Right	Left	Bilateral	105	38	11	Recovered	Died	116		9
							58	49	18	3	13	8	10				3	0		Meningitis	Other causes
																				1	8

of the sinus were involved. Very few among them had extensions towards the base of the skull in the anterior fossa, and a few had an involvement of the outer walls of the sinus only. (See Table I.) Three showed definite cloudiness in the sinus, probably indicative of intrasinus bleeding. All three recovered without operative intervention. In this series, there was no evidence of aërocele involving the sinuses. Dandy (1926),[1] in his thorough article on pneumocephalus, states that a great many are caused by fractures in the region of the paranasal sinuses. These are definitely serious and the mortality, according to this author, is around 50 per cent., due usually to meningitis. One of the evidences of aërocele is the presence of emphysema and crepitation on palpation in the neighborhood.

Results.—Of the total group of one hundred twenty-five, one hundred and sixteen left the hospital recovered. These patients were not followed after their discharge and it is possible that a few of them may have had later complications. During their stay in the hospital, they were free of increase in temperature and all evidences of infection. Nine died. In one, the death was due to chronic alcoholism with pneumonia; the second died of associated brain injury; a third had severe brain injuries and internal injuries; a fourth died of severe brain injury having cerebrospinal fluid leakage from the nose but no evidence of meningitis. Another died of other causes than the brain involvement, namely, fractures of the femur, tibia, fibula, metacarpals, radius, ulna and internal injuries. One died of associated rupture of the bladder; one died of severe injuries in other parts of the body and one died of meningitis. This patient had evidences of bleeding from the nose on entrance. There was no cerebrospinal rhinorrhœa. In these cases emissary veins at the base probably have a great deal to do with the genesis of meningitis. It may be seen that the incidence of meningitis is very low, namely, eight-tenths of 1 per cent.

Operation.—The three cases of compound fracture involving the sinus region were operated on, a thorough débridement was performed, the sinuses cleaned out and packed with iodoform gauze, this being removed gradually in the course of four or five days. All of these cases recovered. It is our belief that compound fractures in this situation should be taken to the operating room as soon as the patient's condition permits and a very thorough débridement and removal of all foreign bodies performed. The one shortcoming of a thorough operation in this situation is the æsthetic result. With a complete Killian operation, patients are definitely deformed and it may be necessary in due course of time to resort to osteoperiosteal grafts to improve their appearance.

Treatment.—It is our belief that in the majority of the cases, the treatment par excellence is conservative. Operative intervention has many drawbacks. First, such operative intervention is not needed in order to curb the incidence of meningitis, for there are very few among them who develop this disease. Second, the ensuing deformities following a sinus operation leave the patient with a psychic problem which is important to keep in mind.

Third, any operative procedure does shock the patient and might accentuate the signs and symptoms of brain injury. For methodical purposes, the treatment of the various types of fracture in this situation will be taken up.

Simple Fractures.—Here the treatment is essentially conservative. The patient's temperature and the possible onset of purulent changes in the sinus should be observed. The appearance of emphysema and crepitation in the sinus region and the occurrence of cerebrospinal rhinorrhœa should be watched for. Granted the patient does not show any of these complications, he is left alone and the recovery is usually uneventful.

Simple Depressed Fractures.—Here again, conservative treatment is advisable unless guided by the clinical condition of the patient, for the majority do well on conservative treatment. To perform radical operations in such cases leads to deformities of the forehead. In case the sinuses are cloudy due to intrasinus bleeding, the patient should be doubly watched for a possible infective process. Three cases of cloudy sinuses in this series recovered with no untoward symptoms.

Compound Comminuted Fractures.—The treatment in these cases is operative. The patient should be taken care of as soon as his condition permits. The initial cut may be enlarged, a thorough débridement and removal of all foreign bodies accomplished, and, if necessary, a complete Killian operation performed. The posterior wall of the sinus should be inspected for fracture, depressions and foreign bodies. It is advisable in some cases to expose the dura and pack. In this series, the three compound comminuted fractures were operated on with good results.

Rhinorrhœa associated with frontal sinus is uncommon. In this series there are only two cases. Fractures of the base of the anterior fossa are more frequently associated with cerebrospinal leakage. In case the frontal lesion is associated with rhinorrhœa, the treatment according to several authors is operative. Among others, Peet, Andruss and Teachenor profess that they be operated on. Peet, in particular, stresses the placing of a gauze pack over the break in the dura, holding it tightly against the brain. Where possible, the tear in the dura should be repaired. Of necessity, such operations are quite shocking and should be performed only if the condition of the patient permits. It should be remembered that a great many of these cases recover spontaneously. In Teachenor's series No. 1, the only patient with rhinorrhœa recovered, whereas the remaining five died of meningitis, brain injury, brain abscess and did not have cerebrospinal leakage. It is essential to get the point of view of the otolaryngologist in this respect. It is known that operations on the paranasal sinuses may be associated with cerebrospinal rhinorrhœa. Campbell, for instance, reports nine cases with five recoveries out of eight. Bromberg[7] reports a case of rhinorrhœa with pneumocephalus secondary to skull fracture who was conservatively treated, being confined to bed for several weeks. This patient recovered Although the incidence of meningitis in such cases is quite high there are many who

recover. By using prophylactic measures, such as abstaining from blowing the nose, leaving the nasal cavities alone and avoiding intra-nasal douches, a great many of these cases recover. Cerebrospinal rhinorrhœa may be associated with penumocephalus. This is a serious complication, the mortality in untreated cases, according to Dandy, being around 50 per cent. Dandy's suggestion that such cases be operated on by means of a frontal flap and repairing the area of leakage seems to be good surgery. However, the necessity for this surgical intervention should be guided by the course of the patient's condition. In this series, we have had no case of aërocele of the frontal sinus. Should they occur and are uncomplicated, they should be left alone. One complication, according to Robb (personal communication) is emphysema of the surrounding tissues with ensuing cellulitis. In such cases, this observer thinks that the frontal sinus should be opened in order to prevent the possibility of septicæmia and meningitis.

Suppurative Sinusitis.—Although no case of suppurative sinusitis following fracture occurred in this series, such a possibility exists and these cases should be watched carefully and very often treated surgically. Suppuration in this situation may be followed by extradural or intradural abscesses or brain abscesses. The patient should be watched for onset of such complications. The face-down position may afford gravity drainage in cases of suppurative sinusitis. With such a position, extension of the infection toward the cerebrum may possibly be curbed.

In summing it may be stated that the treatment of fractures in the frontal sinus region, with the exception of compound fractures, is essentially conservative. Most cases of rhinorrhœa get well spontaneously and even with this complication, we should not find fault with conservative treatment. Infection in the frontal sinus should be carefully watched for and surrounding cellulitis and evidences of infection often call for surgical treatment.

SUMMARY

The incidence of frontal sinus fractures in a series of over 2,600 cases of skull fracture is around 5 per cent.

The majority of sinus fracture cases are asymptomatic. They should be confined to bed for a period of eight to ten days at least. They should not be permitted to blow the nose. Intranasal douches are counterindicated.

The patient should be watched for infection in the sinus and suppuration within the cranial cavity.

Compound fractures in this situation should be operated on as soon as the condition of the patient permits. The posterior wall of the sinus should be inspected.

The great majority of frontal sinus fractures should be left alone. The results with conservative treatment are gratifying.

The incidence of meningitis in this series is eight-tenths of 1 per cent.

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OSTEOMYELITIS OF THE JAWS IN NURSLINGS AND INFANTS

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In another communication, the general subject of osteomyelitis of the jaws was extensively discussed. The subject was found to be a very complex one; and it was pointed out that cases of osteomyelitis of the jaws occurring in nurslings and infants have, because of their extraordinary manifestations, become separated from the general subject of osteomyelitis of the jaws, and have been considered more or less as a distinct entity. The group has been surrounded with several theories as to the pathogenesis of the disease which have little more to substantiate them than a collection of casual and isolated case reports called from the literature and fortified by the analytical consideration of the given writer. Up to the present time this has necessarily had to be so because the disease being a rather uncommon one, it does not fall to the lot of any individual to observe for himself any considerable number of cases.

The present communication deals with such cases of acute osteomyelitis of the jaws—both upper and lower—which occur most commonly in the first few weeks or months of life, and very rarely beyond that period, and which are characterized (1) by pathological manifestations associated with the osteomyelitis which are referable to the mouth, the nose, the nasopharynx and the orbit; (2) by the clinical manifestations associated with an acute infection of severe intensity; (3) by sequestration and loss of the entire jaw and of the teeth which it customarily carries; (4) by the subsequent deformity associated with this loss in the fortunate cases which recover, and (5) by a high mortality.

Similar clinical entities have been described under various names: gangrenous or sequestrating inflammations of the tooth-pulp of early infancy (Bronner); maxillary osteomyelitis of infants (Bronner); gangrenous osteogingivitis (Comby, Cozzolino, Bindi); phlegmonous pulpitis (Bronner); sequestrating inflammation of upper jaw (Van Gilse); peri-alveolar abscess (Moser, S. Kakals); ulceromembranous stomatitis (Gilberti); the stomatitis of Vincent; the sequestrierende Zahnkeimentzundung (Zarfl); and empyema of the Antrum of Highmore in infants (Kelly). The affection of such varied terminology is always described as having a local character; but the confusion in which the subject exists is well illustrated by the fact that at times the cases are described as occurring during the course and apparently as a consequence of some general infectious disease, such as measles, whooping cough, *etc.* Such cases, however, are better dissociated from the group occurring in nurslings and young infants, and are better considered as being ordinary cases of hæmatogenous infection of the upper or lower jaw.

Literature.—An extensive literature has grown up around this subject. The first case of osteomyelitis of the superior maxilla in infants reported in the British literature was by Douglas, in the *British Medical Journal*, in 1898. The first case reported in American literature was by Posey, of Philadelphia, in 1912, in the *Journal of the American Medical Association.*

Clinical observations have been made by Moser, S. Kakals (1899), Comby (1904), Kelly (1904), Broca (1904), Movestin (1905), Cozzoloino (1906), Gilberti (1907), Scott-Ridell (1909), Landwerhammer (1909), Fliess (1912), Rocher (1912), Bindi (1912), Zarfl (1913), J. Francasis (1914), R. Petit (1915), and by Landette (1916). The observation is made by many of these that osteomyelitic and necrotic processes of the jaw-bones are peculiar to the first year of life.

Neumark (1897) tabulated thirty cases of osteomyelitis and finds that the superior maxilla was attacked in three cases and the mandibula in two cases.

Dependorf (1907), from the Surgical Clinic of Jena, reports 600 variously localized cases of osteomyelitis and finds that the process is localized eight times in the mandibula and only twice in the superior maxilla.

In 1922 Marx, in the *British Journal of Ophthalmology*, reviewed the thirty-five cases reported up to that time, paying especial attention to the orbital symptoms. He added three cases of his own. Since that time there have been numerous cases reported both in the American and foreign literature, and it began to be realized that cases of this type are not so rare as it was at one time thought.

Kelly (1924) reported a case of his own to which he added seventeen cases collected from the literature.

Bronner (1925) cites altogether forty cases which he was able to find in the literature, which he regards as maxillary osteomyelitis of infants, but which were recorded under the most varied terms and brings the clinical manifestations into accord with the pathologico-anatomical findings, which he explains.

Bass (1928) reported several cases and reviews the subject from the standpoint of his own cases and from what he gathered from the literature. He added five cases from the literature to those collected by Kelly and quotes a case described by Mayer.

The monograph of Waton and Aimes (1912) contains twenty-three cases of osteomyelitis of the jaws in children between two and thirteen years of age. The report of Cavina-Pratesi (1924) compiled from the Pediatric Clinic in Florence also concerns children from six to eight years of age. These reports had better be excluded from the present discussion.

Etiology.—As regards the etiology, practically all writers are agreed that osteomyelitis of the jaws just as that of the long bones, must be attributed to bacterial infection. As to the nature of the organisms which cause this disease we know but little. The experience of Allard and Sicard is that in the osteomyelitis of infants and young children pneumococci and streptococci

play a much greater and commoner rôle than staphylococci; in older children and in patients of more mature age the latter organisms are most common in cases of bone infection. A coliform bacillus, however, is a common finding in this group of cases. My own experience is that staphylococcus aureus is the commonest organism found in osteomyelitis.

The available sources of the infecting organisms are (1) the vaginal canal of the mother; (2) the fingers of the accoucheur or of the nurse; (3) the nipples and breasts of the mother; (4) the fingers or apparatus used in cleansing the baby's mouth after birth.

Pathogenesis.—The question as to whether this peculiar disease in nurslings is a metastatic lesion similar to other forms of osteomyelitis, or whether it is a primary infection of the jaw and a primary lesion has not received a unanimity of opinion. Only a minority of the observers have taken it for granted that a general infection must preëxist and that the jaw infection is secondary thereto.

Galli reports a case in which he believes that the osteomyelitis of the maxillary bones must be regarded as hæmatogenous in origin and not caused by external affections: a septicæmia was caused by the *Bacillus coli* and in its course it implicated the bone and brought about the renal and hepato-biliary complications. In one of Bass's cases staphylococcus aureus was found in the blood which would also favor the mechanism of a hæmatogenous infection.

The majority of observers have believed that this form of osteomyelitis of the jaws in nurslings and infants is a primary lesion. The various discussions have centred themselves about the method of and the point of entry of the organisms into the maxilla and from whence they came.

Most of the observers who report on this subject are of the opinion that the alveolar border is the spot where the bacteria enter. This supposition appears to them to be perfectly feasible, because this area of the mouth offers the best opportunity for the invasion of bacilli through small superficial abrasions. It is obvious to them that the alveolar process must be the most easily wounded spot in the maxilla.

The small wounds in the mucous membrane may originate in a birth trauma, *i.e.*, pressure produced by a narrow pelvis, or by pressure from the forceps, or by a face delivery where the fingers of the accoucheur must often go into the mouth. Landwehrmann has, moreover, drawn attention to the fact that in cleansing the mouth, small wounds may be inflicted.

According to Bass and others, some have apparently followed infection developing in the mother. According to Marx there is one clear example of infection from the mother's vagina in the literature, and there are cases of infection in nursing infants described by Douglas and Lacasse and by Marx in which a lymphangitis of the breast was present in the nursing mother. Nevertheless, the bacteriological studies in these cases seem to be insufficient. Intrapartum intranasal infection has been mentioned, but this is assumed to be unlikely. In one of Bass's cases the infant was apparently in excellent health, and the labor had been normal. In another of his cases there was a

furuncle on the ankle, and impetigo neonatorum had been present for two weeks. Small skin wounds and ecchymoses were present also in the cases of Wood and Dujardin.

There is a difference of opinion among the various observers who believe in the "primary" nature of this disease as to the part of the jaw—and this applies especially to the upper jaw—in which the infection originates and centres, and from which it spreads. Opinions differ and vary between the nasal cavity, the antrum of Highmore and the unerupted teeth.

Intrapartum intranasal infection has been mentioned. Paunz, of Budapest, claims that all cases are due to an antrum infection, and a minority agree with that position. However, most of the observers agreed that until more detailed investigations of the antrum early in the disease could be done both by X-ray and by puncture with washing, the differentiation cannot be definitely made.

Inflammation of the tooth-buds in the course of a sepsus or an erysipelas in the new-born has been described by Swoboda and by Zwarfl. The last observer made a detailed histological study of such cases and apparently proved to his own satisfaction that a gangrenous process arose in the undeveloped teeth. Finkelstein and von Reuss also believed that osteomyelitis of either of the jaws begins about the tooth buds. The Koerner Clinic, which seems to have investigated these cases closely, hold to the view that it is caused by an infection of the unerupted teeth chiefly the tooth anlagen of the canines and milk molars and with that view the majority of old and new writers on the subject agree.

Proceeding further from these premises the various observers agree that having once settled in the maxilla the bacteria develop further in the spongiosa of the bone. The process increases progressively just as in the osteomyelitis of the long bones; it is followed by suppuration and finally by necrosis and sequestrum formation. Although the clinical picture is sharply defined, its diverse manifestations have caused many of the cases to be described as infections of the antrum of Highmore, others as osteomyelitis of the jaw and others as phlegmons of the orbit. Schmiegelow was the first to show that these cases were in reality cases of osteomyelitis, and Kelly pointed out that the whole superior maxilla is involved, and that evidence of its disease is presented by each of the bony surfaces—the orbital, nasal, facial, and palatal.

My own impression and belief is that osteomyelitis of this type occurring in nurslings and infants is in no way different from the hæmatogenous form of osteomyelitis in general occurring in other parts and bones of the body; and that the occurrence of the lesion in such young subjects is associated with the physical conditions of childbirth and the environmental conditions immediately following. The localization in the jaws is, as is held by almost everybody, due to various forms of minor or major injury received during childbirth or to various forms of trauma received thereafter associated with the care of the child, especially with the cleansing of the mouth. The predilection of the upper jaw is due to its larger size and its more rigid construction

and attachment in the skull which favor the more frequent reception of traumatisms. The presence of unerupted teeth and teeth germs or buds determines areas of more marked vascularity which help to determine points of fixation for the metastatic infection. The traumas described as occurring on the alveolar border or elsewhere, which are taken to be the primary points of entry for the infection, should not be assumed to make pathways directly to the bone; these are points of entry pure and simple and the transmission into the substance of the jaws is only made possible by way of the blood-stream and not by simple extension by continguity.

The mechanism of the pathogenesis and pathology of hæmatogenous osteomyelitis in general has been discussed very extensively on a number of previous occasions and an extensive discussion of this subject will not be repeated here. Suffice to say in résumé that:

Acute hæmatogenous osteomyelitis is a metastatic lesion during the course of a bacteriæmia, the latter resulting from an acute bacterial lesion on a surface of the body which forms the portal of entry for the infection. In this conception a surface of the body includes not only the skin, but also the entire mucous membrane of the alimentary tract, the genito-urinary tract, *etc.* The common surface lesions include not only furuncles, carbuncles, *etc.*, on the skin but also easily demonstrable lesions in the tonsils, and in other lymphadenoid collections lying in the mucous membrane of the pharynx, as well as less demonstrable lesions, such as those in the Peyer's patches.

The fundamental cause of the spreading of the original lesion in the form of metastatic or subsidiary lesions is an infected thrombus lying in the original area of infection, and communicating at some point with the freely circulating blood. Organisms, growing on the surfaces of the thrombus are discharged, or the pieces of the thrombus itself break off and are discharged into the circulation and, becoming lodged for various reasons in the vascular network of various parts of the body, give rise to secondary lesion. Bone tissue, because of its peculiarities in vascular structure, seems particularly prone to the blocking of these thrombo-emboli and the susceptibility to this is particularly increased during the period of growth when the individual bones contain well-marked hyperæmic areas at the junction of diaphysis and epiphysis, around centres of ossification, *etc.*

The various accessory causes, such as trauma, that determine the localization of a secondary focus of infection—fixation point—in a given bone, are associated with accidents in the local circulation which facilitate blocking of any bacterial thrombus-embolus. The essential nature of the pathological process that develops at the fixation point is a thrombo-arteritis or thrombophlebitis, and the process in the jaws is exactly similar to that in other bones in which a dominating position is assumed by the secondary vascular thromboses which must necessarily occur in such a pathological lesion. The all-important secondary effect which these thromboses produce are disturbances of essential nutrition which lead to the death of certain bone cells and the consequent necrosis of certain areas of bone tissue.

The actual pathogenesis and pathology is exactly similar to other cases of hæmatogenous osteomyelitis. The point of fixation in the vascular channels of either of the jaws develops into a thrombo-phlebitis. The occlusion of the vascular channel or channels results in the usual deprivation of blood supply and nourishment; and the amount, degree, and character of the resultant necrosis is in direct proportion to the number, size, or importance of the vascular channel occluded and the amount of available collateral circulation.

The blood supply of the superior maxilla is very abundant and is furnished by a number of moderately large vessels (the infra-orbital (Fig. 1), the alveolar, the descending palating, sphenopalatine, the ethmoidal, the frontal, the nasal, and the external maxillary vessels). Practically all of these vessels are derived from the trunk of the internal maxillary artery. The anastomosis is very free and none of the arteries function as end arteries. The various aspects and areas of the maxilla which are supplied by the various vessels is fairly accurately indicated by their descriptive names. Practically the entire segment of the alveolar process is supplied by the alveolar branch of the internal maxillary artery and its continuation as the posterior dental artery.

A periosteal network is practically non-existent; the little which corresponds to this is derived from an abundant network in the mucous membrane covering the alveolar process of the bone. The physiological proof of this deficiency is found in the total absence of any new bone formation after disease or destruction of any part of the bone.

The vascular arrangement for the inferior maxilla is as follows: The inferior dental artery penetrates the foramen on the inner side of the ramus of the jaw, and runs along the dental canal in the substance of the bone. Opposite the first bicuspid tooth it divides into two branches, incisor and mental; the former is continued forward beneath the incisor teeth as far as the symphysis, where it anastomoses with the artery of the opposite side; the mental branch escapes at the mental foramen, and anastomoses with the submental, inferior labial, and inferior coronary arteries. The dental and incisor arteries during their course through the substance of the bone give off a few twigs which are lost in the cancellous tissue, and a series of branches which correspond in number to the roots of the teeth; these enter the minute apertures at the extremities of the fangs and supply the pulp of the teeth. Collateral circulation is furnished from the opposite artery and by its mental branch with the submental, inferior labial and inferior coronary arteries.

For our purpose this description may be paraphrased as follows: Each inferior dental artery acts as a nutrient artery for its appropriate half of the bone; it perforates the substance of the bone on the inner surface of the ascending ramus of its appropriate side, divides dichotomously and nourishes the bone up to the upper ends of the rami, in fact the two inferior dental arteries are the nutrient arteries of the inferior maxilla. The upper ends of the rami are supplied from adjacent muscular branches.

The periosteal circulation is derived from numerous arterial trunks—muscular and other—in the immediate neighborhood; it is most abundant over all parts of the bone except the upper part of the ascending ramus and

FIG. 1.—To show the blood supply of the superior and inferior maxillæ. The entire blood supply of the superior maxilla is derived from one large arterial trunk, the internal maxillary artery, and the branches supplying the bone consist of, and are arranged, as a number of loops thrown around the bone at different levels and in different planes. For example, note the loop formed by the infra-orbital branch and the posterior dental branch by the interposition of the anterior dental branch of the former, *etc.* The inferior maxilla is supplied by one loop formed by the two inferior dental arteries each of which is also a branch of the main trunk of the internal maxillary artery. IMA—the internal maxillary artery. PA—the posterior alveolar artery. PD—the posterior dental artery. G—the gingival branch of the posterior dental artery. AD—the anterior dental artery. AAL—the vascular loop from which the apical arteries are derived. IO—the infra-orbital artery. ID—the inferior dental artery. M—the mental branch of the inferior dental artery. (After Deaver.)

its coronoid and condyloid processes. Here the periosteal structure disappears in the intimate rugged attachment of muscles, tendons and ligaments.

The interesting part of the blood-vessel arrangement for both the superior and inferior maxillæ is found in the dominating fact that the entire supply is derived from one large arterial trunk, the internal maxillary artery, and that the branches which form the network of supply are arranged in loops based on the trunk of the internal maxillary artery; each superior maxillary bone is fed by a series of loops which are united in a stem based on the trunk of the internal maxillary artery of its appropriate side; the inferior maxilla is fed by a single vascular loop passing from the internal maxillary artery of one side to that of the other. Study of Fig. 1 will amply demonstrate this fact; as an example, note the loop formed by the infra-orbital artery and the posterior dental artery by the interposition of the anterior dental branch of the former; or the loops formed by the gingival branch of the posterior dental artery with the nasal branch of the infra-orbital. It is as if a series of loops had their free ends gathered in a single bundle corresponding to the main trunk of the internal maxillary artery.

This simple anatomical fact explains very adequately all the clinical forms of osteomyelitis of the jaws which occur in this group of cases as follows:

(1) Involvement of the entire bone with manifestations referable to the palatal, nasal, and orbital surfaces results from a lesion in the stem of the internal maxillary artery prior to the giving off of the posterior alveolar branch with or without extension thrombosis in the loops described above which are derived from the internal maxillary artery. Collateral circulation is at a minimum and a maximum lesion results.

(2) Involvement of the alveolar process results from a lesion in the course of the posterior dental and gingival arteries. The amount of bone involved depends on the amount of collateral circulation.

(3) Involvement of the anterior part of the alveolar process and the adjacent part of the bone results from a lesion in the course of the junction of the posterior and anterior dental arteries.

Involvement of the antrum of Highmore occurs with either Group 2 or 3. The amount of bone involvement depends on the possibilities of collateral circulation.

(4) Involvement of the palatal surface results from a lesion in the palatal arteries or may be an extension from an involvement of the alveolar process.

(5) Involvement of the nasal aspects of the bone results from a lesion in the nasal and anterior branches of the infra-orbital artery. This variety may also be associated with an empyema of the antrum of Highmore.

(6) Involvement of the orbital aspect of the bone results from a lesion in the course of the infra-orbital artery.

The controversy as to whether this form of osteomyelitis of the jaw centres in and is derived from a lesion in the teeth buds, the nose, or the antrum, or in the neighborhood of the orbit is to my mind unnecessary and fruitless, inasmuch as from the facts outlined, one can easily see that all of these manifestations are simply determined by the dominating position of the thrombo-phlebitis in the course of the vascular channels described. The

seeming importance of any one manifestation is only a superficial one and the character of the pathogenesis and pathology is, as in other forms of acute osteomyelitis, intimately related with the position of the thrombo-phlebitis and the resultant necrosis.

General clinical course.—In 82.5 per cent. of the cases reported in the literature the first two months of life were involved, the greatest number of the cases falling within the second and third week. With few exceptions, the superior maxilla was most frequently diseased. The right and left side were about equally involved.

The disease presents a clear-cut definite picture, one case report being very much similar to the other. The disease usually befalls healthy infants between two and ten weeks of age, sometimes older ones. Prodromal periods differ, sometimes the children cry and refuse nourishment, and there may or may not be a slight rise in temperature accompanied or not by diarrhœa or constipation; in other cases the children are violently ill, with high temperatures, vomiting, and convulsions.

Then the swelling appears. It may begin in the cheek or in the infra-orbital region, and is almost always accompanied by œdema of the lower eyelid. Sometimes there is exophthalmus from œdema of the orbit. The sclera is inflamed; conjunctivitis is present; and there is sometimes chemosis. It is these symptoms which usually bring the patient to the ophthalmologist; for instance, three out of four of Nord's patients came from the ophthalmologist. In the majority of the cases, a localization forms below the inner canthus of the eye. At this site a swelling in the cheek appears, with redness, abscess formation, the breaking through of pus, and the formation of a fistula. In the mouth can be noticed swelling of the alveolar process and of the hard palate. This may appear even before the swelling beneath the orbit, and is likewise followed by perforation, the discharge of pus, and fistula formation. Numerous small sequestra discharge through the sinuses. Another common and characteristic symptom is the discharge of premature teeth through the alveolar sinuses. In nearly all cases there is sooner or later a discharge of pus from the nose, which is increased by pressure on the abscess. All of this occurs within a few days.

The majority of the children are really sick, but a few remain surprisingly well in spite of the progress of the disease. The temperature is irregular, convulsions frequently are present, and there is marked anorexia and difficulty in nursing, due to the pus in the nostril. The outcome is either healing with or without the persistence of discharging sinuses or the development of secondary purulent foci. Contrary to what one might expect, the prognosis is not always unfavorable. In the fatal cases the infants die as a result of the virulence of the infection before secondary foci have time to develop.

The local manifestations correspond to that of a sequestra-containing abscess of larger or smaller extent. These accumulations of pus correspond in their pathology to subperiosteal abscesses (such as occur in other locations

especially in the long bones) in relation to the appropriate surface of the maxilla. In the upper jaw the abscesses develop as follows:

Usually as a final stage a single large abscess cavity exists which harbors the entire, or the major portion, of the necrotic upper jaw; pus is present on all sides in relation to the palatal, alveolar, nasal, orbital, and retro-maxillary aspects of the jaw. In exceptional instances, owing to the position of the thrombo-phlebitic lesion and to an extraordinary amount of collateral circulation, the process seems limited to only one aspect of the bone; then it either accumulates in relation to the upper aspect of the jaw, in which case the manifestations are mainly orbital; or the process seems more limited to the lower aspects of the jaw and the manifestations appear mostly in the mouth or nose. Frequently the primary accumulation of the pus is in the antrum of Highmore; the abscess then usually points primarily in the nose and secondarily in the alveolar process.

In the average case when a probe is passed into one of the discharging sinuses, it passes at once into a large cavity. This may or may not be a much enlarged antrum cavity; it may be one of the enlarged dental sacs, or it may be a large irregular cavity in which practically the entire superior maxilla is housed as a sequestrum.

Mouth symptoms.—Eighty per cent. of the fistulæ in the mouth are found at the alveolar arch of which more than 50 per cent. are in the region of the canine tooth. In 60 per cent. of the cases an expulsion of the dental pulp occurs. The discharge of teeth and tooth buds through the fistulous openings in the alveolar process, is the most characteristic phenomenon of this disease. It leaves the child without teeth over the affected area. The final result, when the child lives, is a considerable deformity of the face and palate with loss of teeth, both temporary and permanent, on the side involved.

Nasal and antral symptoms.—A purulent discharge from the nares is a very common symptom and a fistula is present in the nose which leads to bared or necrotic bone.

As to the pathogenetical side of the question, Van Gilse says that there has always been an infection of the nose in the cases that he has seen. Kummel, on the other hand, says he has never seen infection of the nose. The nature of the anatomical relationships makes it easily possible for one man to see a whole series of cases with rhinogenous manifestions of an osteomyelitis of the upper jaw and for another to see a series with predominating manifestations of another kind. Sometimes a history of rhinitis is not reported but examination shows pus in the middle meatus. Sometimes it is not possible to detect a preceding rhinitis even when there has been one. In Van Gilse's cases the maxillary sinus was definitely found diseased.

Certainly in an advanced case, and, commonly, in early stages of the disease, manifestations referable to the mouth, the nose, and the antrum of Highmore appear as a single entity. Among the various observers, discussion is frequent as to the relative merit or importance of one, over any, of the other of these localizations. For instance, some observers have held that

large suppurated dental follicles have been mistaken for the small maxillary sinus and a great deal is made of an apparently valid objection that such propagation from the maxillary sinus has never been demonstrated histologically. It is pointed out that in order to accomplish this it would be necessary either to examine cases in which the process is not very far advanced or ones in which the mucous membrane of the maxillary sinus is not involved though the disease of the maxilla is far advanced. However, from the discussion in this paper, it should be obvious to everyone that this differentiation is useless and of no importance either in the understanding of the disease as a whole, or of its individual manifestations.

Eye symptoms.—Of the thirty-five cases tabulated by Marx, thirty developed eye symptoms, and Marx is not certain that the remaining five did not have them. In eight of the records, the eye symptoms first attracted the attention of the patient's family or of the doctor. This is no small percentage, and the eye symptoms are important because it may happen that the ophthalmologist is the first to be consulted, and the further progress of events may depend on his being able to arrive at a correct diagnosis without unreasonable loss of time. This is not always the case and examples of this kind are to be found in one of Marx's cases and in a case of Dujardin.

The eye symptoms which appear in osteomyelitis of the superior maxilla can, for simplicity's sake, be divided into those of the eyelids, of the conjunctiva, and, of the eye socket. The symptoms in the eyelids according to Brown Kelly, are those which first attract attention to the process. The swelling, owing to the severe inflammation of the bone, is a severe collateral œdema, and is quite easy to understand.

The swelling and redness in the region of the lacrymal sac and the subsequent fistula are frequent symptoms. They are important because they may also easily lead to an incorrect diagnosis. According to Brown Kelly an abscess often forms in the lower eyelid, and that pus exudes from the lacrymal sac. François expresses himself more cautiously when writing that the discharge from the fistula in the inner corner of the eye causes one to think of a dacryocystitis, but that the accompanying symptoms at once lead the diagnosis in another direction. The fistula is to be explained by the fact that the inflammation often prematurely develops in the nasal process of the superior maxilla and the pus collects there, eventually seeking the easiest method of exit, which in this case leads to the inner corner of the eye. The canaliculi remains untouched, and it is well to remember this, because one might be too zealous, and, to the patient's detriment, begin an active treatment of the canaliculi if one did not pay proper attention to the other symptoms. Marx advises one to be as conservative as possible and even in the commencement of abscess formation in the lower eyelid to make no incision in the abscess, as frequently treatment via the palate and processus alveolaris will effect a complete cure, whilst an ugly drawn scar with ectropion may result from incision, as appears from the report of Avellis.

The conjunctivitis, like the swelling of the eyelids, is usually part of a

collateral œdema. The degree of severity of the original infection can thereby appear to a certain extent from the chemosis.

Exophthalmus was observed by Marx in ten of the thirty-five cases. In comparison with the other eye symptoms, this appears too seldom to be regarded as an expected phenomenon. Exophthalmus is also an accompaniment of inflammation of the ethmoid. Stephenson has reported ten cases of inflammation of the ethmoid, which all showed exophthalmus to a greater or lesser degree. Thus it appears that the protrusion of the eye is attendant on the inflammation of the bony walls of the orbit fossæ which is followed by purulent exudation in the eye socket.

It is surprising that in the ophthalmic literature, practically nothing is to be found about the ocular complication of osteomyelitis of the upper jaw. There is one reference by Eversbuch (Die Augenerkrankungen im Kindesalter), in which he points out the possibility of mistaking a fistulous osteomyelitis of the superior maxilla for a discharging lachrymal duct.

Differential diagnosis.—A differential diagnosis must be made from the following conditions:

(1) *Ophthalmia neonatorum.*—Usually both eyes are affected, and gonococci can usually be found. Furthermore, the œdema does not extend to the infra-orbital region, nor to the alveolar process. (2) Erysipelas. (3) Dacryocystitis. (4) Syphilis. (5) Tuberculosis.

Careful examination of the patient together with the requisite laboratory work in association with a carefully taken history should result in a correct diagnosis. In addition, the characteristic phenomenon of exfoliation of teeth or tooth buds should by itself suffice to call one's attention to the correct condition.

Blood cultures.—One of Bass's cases had staphylococcus aureus in the blood. The literature otherwise does not contain any specific statements in regard to this aspect of the disease. I feel sure, however, that cultivations of the blood behave no differently in osteomyelitis of the jaws than they do in osteomyelitis of other bones and to this statement Bass's case is a confirmation.

Treatment.—The general principles governing the treatment of acute osteomyelitis in general have been fully described on several previous occasions and will not be repeated here. Suffice to say here that in nurslings and infants osteomyelitis of the jaws should be divided for purposes of treatment into the following two groups:

(1) This group consists of the cases which terminate fatally as a result of the profound toxæmia which develops. The fatalities usually occur in early stages of the disease and whatever one does in the way of treatment is mostly of a temporizing and palliative nature and shows no influence at all on the progression of the local and general phenomena of the disease.

(2) This group contains the remainder of the cases. It is most important to be as conservative as possible. In the early stages, careful cleansing of the mouth, possible poulticing in appropriate cases, and conservative attention to

the nasal and orbital manifestations are all that is required. As soon as definite areas of fluctuation are discovered, these should be adequately but conservatively incised with due attention to the placing of the incisions. As much as possible should be done from the interior of the mouth and through the nasal cavities. It is surprising how one can reach foci of suppuration at apparently distant points from the buccal cavity especially in the interval between the cheek and the bone; especially in the canine fossa this is possibly due to the general involvement of the entire bone in the process. Marx has been able to adequately drain orbital abscesses from appropriately placed incisions in the mouth. There is no record available of any permanent effect upon the eye itself or upon sight.

Sequestration being common, one should assist nature as well as one can in enabling these necrotic fragments to be discharged. The teeth usually are discharged from the fistulæ in the alveolar process; the major portion of the bone itself usually comes away through an opening in the fold between cheek and bone.

Abscesses in association with the lower jaw are more simple technically; and one should follow ordinary surgical principles in treating them. Sequestra should not be removed until involucrum formation is abundant.

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RÖNTGEN VISUALIZATION OF THE PAROTID GLAND BY MEANS OF LIPIODOL INJECTION

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of New York, N. Y.

FROM THE DEPARTMENT OF ROENTGENOLOGY OF BETH ISRAEL HOSPITAL

A review of the literature on the salivary glands shows a paucity of material other than that relating to the subjects of calculus, parotitis, and tumors. The reasons for this are not only the infrequency of other conditions, but also the great difficulty attendant upon the study of changes taking place in the structure of the glands. This difficulty has prompted us to seek a method for visualizing them.

In studying the pathological changes which take place in the gland, the X-ray examination contributes a considerable amount of information not obtained by any other method. Clinical examination, aside from its value in giving information as to size, consistency and mobility, is unsatisfactory and incomplete. Röntgenological examination until recently has been useful only in the study of calculi. Our studies prove the value of the X-ray in demonstrating actual pathological changes in the gland structure.

The technic of injection is simple and, when properly done, is free from injurious after-effects. A blunt, flexible, thin, silver canula; a 5-cubic-centimetre syringe; and a fine, flexible probe with a blunt end are all the instruments necessary.

The lipiodol is first warmed so that it will flow easily. In injecting the parotid duct, the patient is seated with the mouth open, and the operator uses the thumb of his left hand to retract the cheek. The first and second fingers then press the cheek inward in the region of the papilla. A colored solution of some kind, such as 3.5 per cent. iodine, is applied to the papilla, and the opening of the duct is disclosed. With the operator maintaining his position, the flexible probe is inserted and passed into the duct for a short distance in order to verify its patency. In doing this, extreme care must be taken lest the duct be perforated. The probe is then withdrawn, and the flexible canula inserted in its place and from 1 to 2½ cubic centimetres of the warmed lipiodol injected slowly. There will be some external swelling in the parotid region and some slight discomfort. When pain is felt, the injection is discontinued.

The exposures should be made immediately. A 23-degree reverse angle board is used and the position is the same as that for a temporo-mandibular joint or the ascending ramus of the mandible.

In the presence of any active infection, it is not advisable to inject lipiodol on account of the danger of spreading the infection.

In order to intelligently interpret the films, a complete understanding of

the anatomical structure and the pathology of the glands is required. The salivary glands are of the compound racemose type, consisting of many lobes which are made up of smaller lobules, bound together by dense areolar tissue, vessels, and ducts. The individual lobules consist of the ramifications of a single duct, whose branches end in dilated alveoli on which the capillaries are distributed. A basement membrane encloses each alveolus, being continuous with the membrana propria of the duct, and consisting of a network of branched and flattened nucleated cells. Grossly, the parotid gland, which is the largest and most important of the salivary glands, is situated on the side of the face, lying immediately below and in front of the external ear. The gland is superficial, irregularly quadrilateral in form, and is bounded roughly by the mastoid process and sterno-cleido-mastoid muscle posteriorly, and the ramus of the mandible anteriorly. The deep surface of the gland extends inward almost to the pharyngeal wall. A small portion lying immediately below the zygomatic arch is usually detached from the main gland. This is the accessory lobe.

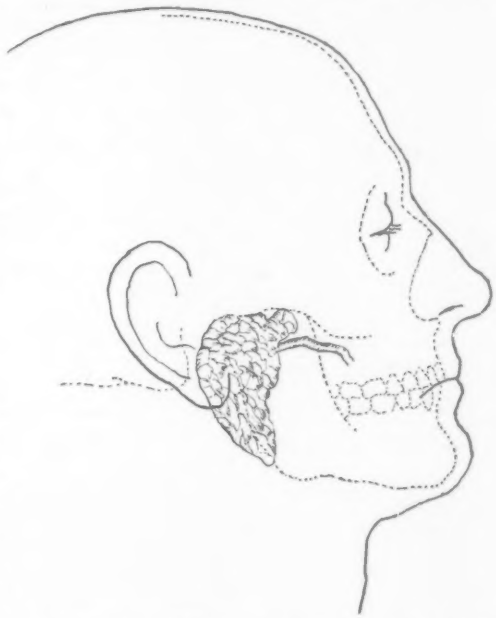

FIG. 1.—Anatomical sketch showing position of the parotid gland.

The ducts of the parotid gland begin within the lobules, and are known as intralobular ducts. Uniting, these run between the lobules and are then known as minor or interlobular ducts. Uniting still further and becoming larger in calibre, as major or interlobar ducts they run between the lobes, and forming numerous branches from the anterior part of the gland, these finally unite to form Stenson's duct, which crosses the masseter muscle at which point it receives the duct of the accessory lobe. At the anterior border of the masseter muscle Stenson's duct turns inward almost at a right angle, and piercing the fat and buccinator muscle, opens on the oral surface of the cheek opposite the second molar tooth. The relation of the parotid gland to the bony structures is shown in the accompanying sketch. (Fig. 1.)

Since it is evident that changes in the gland will manifest themselves by changes in the appearance of the ducts, it will be convenient to refer to the picture of the normal duct markings as possessing a tree-like or arborescent quality.

Fig. 2 shows the normal parotid gland of an adult male. The stream of lipiodol flows along Stenson's duct for a distance of about one-half inch, at which point it is

seen to divide into two branches, one branch going to the accessory lobe, the other continuing to the main gland. As the duct approaches the gland substance it divides into smaller ducts; these major branches extend into the gland between the lobes and divide still further into minor branches which pass between the lobules, and finally into the

FIG. 2.

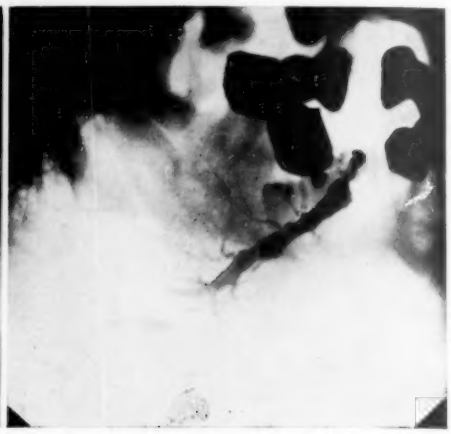

FIG. 3.

FIG. 2.—Normal parotid gland. Male, aged fifty years.
FIG. 3.—Parotid gland two months after clearing up of pyogenic infection and removal of calculus. Male, aged fifty years.

intralobular ducts. The arborescent quality possessed by the normal glands is striking, and gives one the impression of a healthy tree without leaves.

The ordinary conception of Stenson's duct, as a simple tubular structure of even calibre, leading from the gland to the mouth, is incorrect. The duct

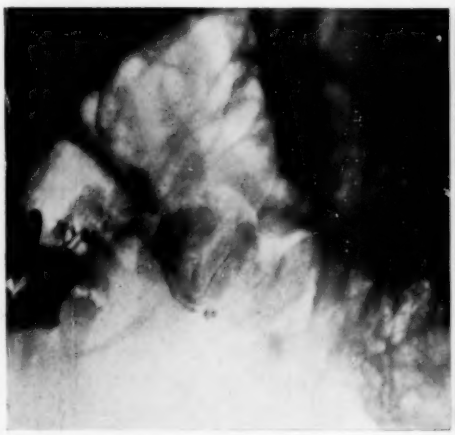

FIG. 4.

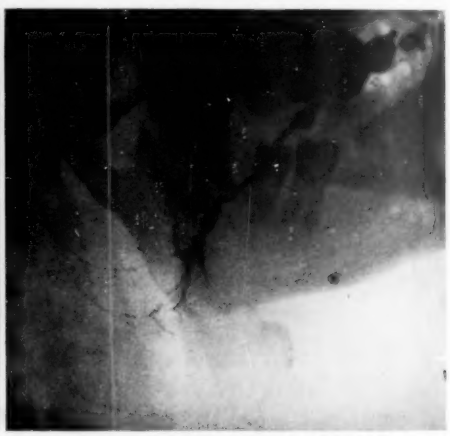

FIG. 5.

FIG. 4.—Parotid tumor of twenty years' duration. Operated upon eighteen years ago. Re-operated upon after this X-ray was taken. Microscopical diagnosis.—Endothelioma. Female, aged fifty-two years.
FIG. 5.—Parotid tumor after course of X-ray treatment. Male, aged forty years

not only varies irregularly in calibre, but is somewhat corkscrew in effect. This serves to explain how the secretion may be stopped by a calculus acting

as a ball valve, the rarity of foreign bodies passing along the duct for any distance, and the difficulty in probing the duct. One must also not overlook the fact that the movements of the cheek affect the duct, straightening its course or making it more irregular.

Fig. 3 shows the parotid gland of a fifty-year-old male, two months after disappearance of a pyogenic infection and removal of a calculus, the history being of eight years' duration. After meatotomy of the duct and removal of the stone, the pyogenic infection cleared up, but only after considerable discharge of pus which indicated a wide destruction of the glandular substance. The back pressure and infection in these cases cause a marked destruction of the acini. This picture was made to determine the condition of the gland two months after the infection had completely disappeared.

Although Stenson's duct is normal in length it is markedly dilated. No accessory lobe is present. Major ducts are present but diminished in number. There are some minor ducts but no intralobular ducts can be seen,

FIG. 6.

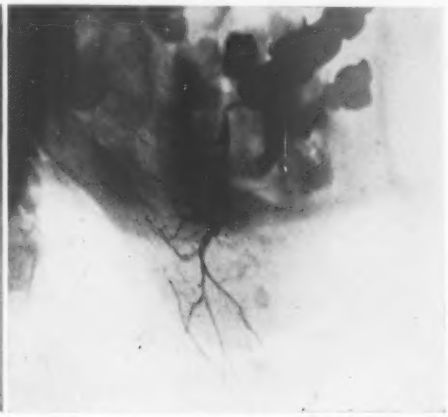

FIG. 7.

FIG. 6.—Parotid tumor involving only the accessory lobe of the gland. Male, aged twenty-four years.
FIG. 7.—Parotid tumor. Duration eight months. Male, aged thirty-nine years.

indicating marked destruction of the secreting portion of the gland. The true arborescent quality is absent, and the appearance suggests a picture of a dead tree.

Fig. 4.—This was a female, aged fifty-two, who gave a history of an operation on the right parotid gland eighteen years ago. At the time of our examination, there was palpable in the region of the gland a firm, rounded, movable tumor about the size of a walnut. There was no pain.

Lipiodol injection shows Stenson's duct to be markedly constricted. It is looped near its orifice like the script letter "e." There are no duct markings as are seen in the normal parotid, but there appears to be a great deal of dense fibrous tissue. The arborescent quality is entirely absent.

The tumor was removed surgically and a pathological diagnosis of endothelioma made.

Fig. 5.—This patient was a male, aged forty, with a parotid tumor that had been treated by X-ray. This picture was taken several months after completion of the course of treatment. Stenson's duct is of normal calibre, but somewhat lengthened; this lengthening may be only apparent due to destruction of major ducts, which are diminished in

number and calibre. Few minor ducts are present and the absence of intralobular ducts points to destruction of the secreting portion of the gland. The arborescent quality is considerably diminished.

Fig. 6.—This was a male, twenty-four years of age, who had a parotid tumor involving only the accessory lobe of the gland. The condition was of one year's duration. Lipiodol injection shows Stenson's duct to be of narrowed calibre and undulating course. The duct of the accessory lobe joins Stenson's duct about one-half inch from the orifice. The duct markings of the main portion of the gland are normal, possessing the arborescent quality. The absence of the duct markings of the accessory lobe indicate a diagnosis of tumor involving only this lobe.

Fig. 7.—This was a male, thirty-nine years of age, who for the past eight months had a mass below and in front of the ear in the region of the angle of the jaw. During the last three months the mass increased in size rather rapidly and there was a moderate amount of pain.

Throughout the entire picture the fine intralobular duct markings are not present. This absence of the true arborescent quality is not due to a deficiency of injection. The

FIG. 8.

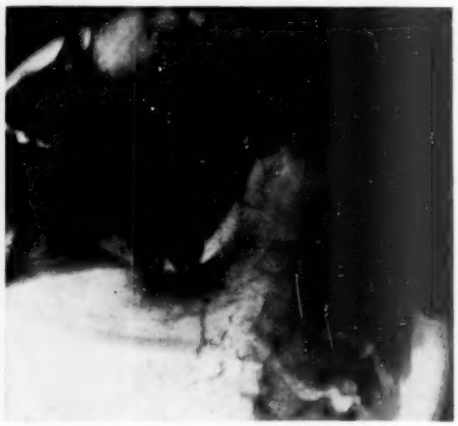

FIG. 9.

FIG. 8.—Parotid tumor. Duration six years. Male, aged forty-eight years.
FIG. 9.—Parotid fistula of twenty-three years' duration. Patient, aged twenty-seven. X-ray with lipiodol injection and stylet inserted into fistulous opening on face. At the age of four years this patient had a swelling on the right side of the face operated upon. Unable to determine the precise nature of the condition for which the operation was performed.

poor definition of the duct markings is noted particularly in the region of the angle of the jaw. We feel that this parotid tumor, on account of its rapid growth, the presence of pain, and the widespread absence of fine duct markings, possesses a malignant character.

Fig. 8. This patient was a male, forty-eight years of age, who had a soft mass in the cheek for six years; the growth was very slow and the patient was practically free from pain during the entire period.

Lipiodol examination shows the retro-angular portion of the gland to be normal, but from this part of the gland up to the point where the ducts unite to form Stenson's duct, the markings are indistinct, and those that do appear are constricted. This area corresponds accurately to the region of the tumor of the cheek, absence of pain and localization of destruction of gland substance is probably a more benign condition.

Fig. 9.—This was a female, twenty-seven years of age, who gave a history of an operation on a swelling on the right side of the face at the age of four years; the precise nature of the condition or operation we were unable to determine. At the time of our examination (twenty-three years after operation) there was present a fistulous open-

ing, pinpoint in size, on the right side of the face about one and a half inches anterior to the external auditory meatus; a fine stylet could be passed into the fistulous tract.

The röntgenogram taken with stylet in place following injection of lipiodol into Stenson's duct shows the fistulous tract opening directly into Stenson's duct near its formation; at this point there is a small triangular cavity filled with lipiodol. In a case of this type examination by lipiodol injection is important as the treatment of a fistula of the duct differs from that of a fistula of the gland proper.

Fig. 10.—This was a female, aged forty-nine, who gave a history of one year's duration of a tumor in the region of the left parotid gland, which had been gradually increasing in size and was accompanied by pain; an operation was performed before an X-ray was taken, and a small encapsulated tumor removed from the body of the parotid gland. Pathological diagnosis of basal-cell epithelioma was made and the patient received a course of X-ray therapy. Three weeks after the last treatment she was referred to us for examination by lipiodol injection.

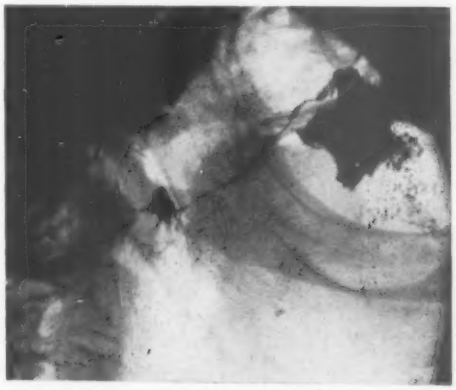

Fig. 10.—Parotid tumor post-operative. Röntgenogram taken three weeks after the removal of a small tumor from the body of the gland.

The röntgenogram shows Stenson's duct to be normal in appearance; near its formation there is an irregularly oval cavity filled with lipiodol, which is probably the area from which the tumor was excised; the arborescent quality is considerably diminished throughout the gland as a result of the radiation therapy. Re-examination with lipiodol injection four weeks later showed the condition unchanged.

CONCLUSIONS

(1) Röntgen examination of the parotid gland after lipiodol injection is a simple, safe procedure when properly performed. In our series no injurious effects have resulted from the injection.

(2) Injection of the gland with lipiodol should never be made in the presence of active infection.

(3) At present, this method is the only accurate way of determining the exact condition of the gland and its deviations from the normal when diseased. In cases of parotid fistula, actual visualization of the ducts is of the greatest aid in determining the course of treatment.

PRIMARY TUBERCULOSIS OF THE PAROTID GLAND

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of Brooklyn, N. Y.

from the cumberland hospital under the auspices of the mountainside hospital research fund

Tuberculosis of the parotid gland, according to medical literature, is rare. We therefore feel justified in reporting the following case:

A. D., aged forty-eight, presented herself October 5, 1930, with a swelling of the right parotid gland which had come on gradually during the previous ten months. There had been no pain until two months previous when she began to experience neuralgic pains along the right zygomatic region. The mass was firm in consistency, the skin was glazed, reddish and tense, but not attached to the mass. The tumefaction extended upwards to the zygoma, downwards over the ramus of the mandible and the angle of the jaw anteriorly to the angle of the mouth. A thorough physical examination was negative. Wassermann and other tests were also negative. There were no cervical glands palpable. The pre-operative diagnosis rested between mixed tumor of the parotid, sarcoma, and tuberculosis, the first seeming the most likely because of the slow growth and duration. Tuberculosis was not considered because of its rarity.

October 20 operation for removal was done. By an incision starting at the angle of the jaw and running along the ramus of the mandible, and another from the angle upwards to the zygoma, the tumor was exposed. All was removed except a small portion that was closely adherent to the main trunk of the facial nerve. The post-operative career was uneventful except for a salivary fistula that formed at the lower incision which cleared up at the end of two weeks. The patient was then given two X-ray treatments and has now made a complete recovery.

The pathological report submitted was as follows: These sections show a predominance of epithelioid tubercles throughout, with central areas of organization and giant cells of the Langerhans type with an outer zone of fibrous connective tissue and round cells. This is a rare condition of the parotid gland. It is advisable that a thorough examination be made for the primary focus of tuberculosis in this case.

Diagnosis.—Tuberculosis of the parotid gland.

Review of Literature.—The parotid gland may be infected with tubercle bacilli either through Steno's duct by way of the lymph-channels or through the blood-vessels.

In October, 1914, Thomas F. Carmody gave a review in the Laryngoscope. He reviewed the cases of tuberculosis of the parotid gland, of which, at that time, there were four in this country and eleven abroad. He mentioned such cases as de Paoli's case, which was a male aged thirty-three, without a previous personal or hereditary history of tuberculosis, who presented himself with a swelling of the left parotid about the size of a hen's egg, a size attained in six months' time. He further presented a facial paralysis on the affected side. Under diagnosis of sarcoma the tumor was removed. Examination revealed tuberculosis. This author ventured no opinion as to the origin of the tumefaction.

In 1894, von Stubenrauch reported a case which presented some different characteristics. A male, aged sixty, who had suffered from stomatitis and accompanying salivation, presented a small fluctuating mass in the right parotid region, which was very painful. *Pathological examination.*—A large pseudo-cyst with liquid contents similar to saliva, and with a wall about two centimetres thick internally showed tubercular granulations, while externally there was a capsule consisting of fibrous connective tissue, with

the remains of excretory ducts, led von Stubenrauch to believe that infection took place through Steno's duct.

In 1895, another case was reported by de Paoli. The patient was a girl of nineteen, whose left parotid was involved, and in spite of his previous experience, he made a diagnosis of fibro-sarcoma. Pathological examination after removal proved it to be tuberculosis. In the same year Legueu and Marien reported a case of a girl of thirteen years, in whom there had formed during a long period a swelling as large as a nut, covered with skin, and apparently adherent to the left parotid gland. A diagnosis of adenitis was made, but examination disclosed many tubercles diffused in the parenchyma of the gland, and a central softened mass. The case of Bockhorn, reported in 1897, was that of a lady, thirty-nine years old, with no previous history of tuberculosis. There was to be seen a swelling of soft consistency, which had formed in the left parotid region in the previous three months. Pathologically it was recognized as tuberculosis of prob-

FIG. 1.

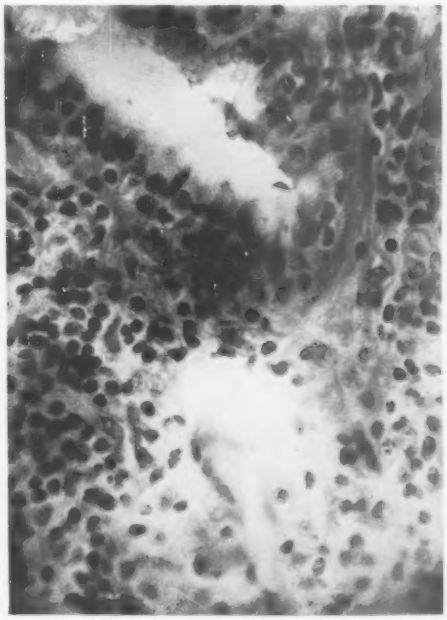

FIG. 2.

able vascular origin, on account of the great abundance of tubercles in the region of the vessels. The pre-operative diagnosis was a syphilitic gumma.

In 1897, Kiesow published at *Griefswald* "Ueber einen Fall von Isolierter symmatrisher Tuberkulose der Parotis" (Case of Isolated Symmetrical Tuberculosis of the Parotid Gland). A girl of thirteen had for three years presented swelling in both cheeks, about the size of a pigeon's egg when first seen, not painful, and giving the appearance of an attack of mumps. The patient was undersized and mentally retarded. Excision was attempted under diagnosis of sarcoma but histological examination showed early tuberculosis.

Parent's patient reported in 1898 was a man of sixty-one with a previous right otitis media and dental caries. A painful swelling had been present in the left parotid region for three months. A mixed tumor was diagnosed and the swelling removed. The immediate result was left facial paralysis. Pathological examination of the specimen showed the characteristics of tuberculosis, probably of hematogenous origin, because, as in Bockhorn's case, the distribution of the tubercles corresponded with the vessels.

Recovery was complete. Tuberculosis granulation and tubercle bacilli with caseation were found by Lacent in a young man of twenty-nine, with a family history of tubercular infection. Mintz reported, first in Russian and later in a German translation (Deut. Zeits. f. Chir., vol. lxi, p. 290, 1901), a case of a three-year-old-child. A lesion in the left parotid gland proved on excision and examination of a tissue specimen to be tuberculous, all stages of inflammation and caseation being in evidence.

Scheib's case, reported to the German Pathological Society in 1900, was that of a girl of fifteen, who had a swelling in the right cheek which had been painful at night for a fortnight. There was cough with expectoration, loss of appetite and night sweats; temperature 38.1° C. At the opening of Steno's duct was a red protuberance the size of a small pea. The tumor was soft in its centre, and upon being incised exuded pus in which tubercle bacilli were demonstrated. The child died a month later of pulmonary tuberculosis. Post-mortem examination showed thrombosed vessels in the region of the abscess, which led the author to believe the infection to be hematogenous.

American cases seen up to the time this article was written were those of Frank and Wood, which are separately abstracted, and of C. L. Scudder. Scudder's patient was a middle-aged woman, who had noticed a small swelling below the lobule of the right ear of five years' duration. When seen it had reached the dimension of two inches in diameter, being hard and rounded with irregular indurated borders. There was no pain or tenderness on palpation. The mass was removed, with division of Steno's duct an the facial nerve, as these were involved in the disease process. A year and a half later there was no recurrence but facial paralysis was present on the involved side. Microscopical examination of the removed specimen showed numerous small foci in the gland. These were composed of epithelioid, small, round and numerous giant cells, with cheesy degeneration. *Diagnosis.*—Tuberculosis. Further cases were published by Borchardt in 1903, Cole in 1904, Püppel in 1905, Danielson in 1907, Fiorvanti in 1910, Nadel and Pouget in 1911.

Carmody's case was a patient at the National Jewish Hospital in Denver, a male aged twenty-four, born in Russia. When first seen there was a hard swelling of the left parotid gland which was diagnosed as inflammatory parotitis due to trauma. This was opened and drained, a few diplococci being found in the evacuations. In dressing this wound another abscess was found walled off from the first, in which pus, containing tubercle bacilli, was found. Soon after there was rapid swelling of the right parotid, and when this was opened, tubercle bacilli were found in the discharge from the wound. The diplococci also present were found to be identical with the organisms causing parotitis. The right side healed completely, but on the left a fistula remained for the two years elapsing before death occurred from pulmonary tuberculosis. No facial paralysis occurred although nearly the entire left parotid gland sloughed away. There was a slight drooping of the right eyelid, but this quickly disappeared.

J. L. Emerit reports a "New Case of Primary Tuberculosis of the Parotid," These de Paris, 1923. This thesis does not add anything to the facts set down in the papers previously abstracted. The cases cited are given in greater detail, but nothing concerning pathogenesis nor treatment has been added. The patient was a woman of thirty, who had been aware of enlarged but painless glands in the region of the right parotid for many years. There was a history of excessive cough, and recent loss of weight preceding the manifestation of active symptoms in the swollen parotid region. Excision was undertaken and the specimen removed was histologically proven to be tuberculous. The patient made a good operative recovery and when last heard from had had no recurrence and showed general systemic improvement.

L. Haslhofer, in Virchow's Arch. F. path. Anat. u. Physiol., vol. cclxvi, p. 499, 1927–1928, reports a case where the patient was a woman of forty-five, who for six years had been conscious of a swelling in the left parotid region. It had only recently become painful, for which reason she sought medical advice. There was a history of spondylitis

in childhood and a fall upon the hip at the age of thirteen, since when there had been stiffness in the hip-joint. Goitre was present. A fluctuating tumor was palpable in the left parotid region. This was excised, the patient making a satisfactory operative recovery.

H. M. Cunningham (Vancouver, S. C.) reports a case of tuberculous parotitis in laryngoscope, vol. xl, p. 116, February, 1930. A girl, aged eleven years, presented a mass in the left parotid region; no pain nor tenderness; overlying skin freely movable. The mass extended from a point downward to the ramus of the jaw and backwards behind the lobule of the ear.

Under diagnosis of probable sarcoma, excision was attempted. As this was found impossible without sacrifice of the facial nerve, a frozen section was made and the procedure halted. Report on the frozen section was "Mixed Tumor." As the condition was of brief duration, and clinically there was much evidence of inflammation, the wound was temporarily closed and a more accurate examination demanded.

The second report was: "Section through the piece of tissue shows complete replacement of the glandular elements, a well-defined granulation process consisting of fairly numerous tubercle formations composed of epithelioid and giant cells, these tubercles occasionally undergoing caseation, but not to any marked degree, the whole surrounded by rather dense fibrous connective tissue richly infiltrated with small round cells." *Diagnosis.*—Tuberculosis parotitis.

Under quartz light treatment, the child's condition improved, the tumor disappearing entirely. After three years there had been no indication of recurrence. The outstanding features of this case were the rarity of the condition, the futility of depending too much on diagnosis from frozen sections, and the remarkable efficacy of the quartz light treatments.

The recognized avenues of infection of tuberculosis of the parotid are hematogenous, lymphatic and canalicular. Lymphatic infection and hematogenous infection are hard to differentiate on account of interlacing of the vessels, a point mentioned by Nadel and Puget. Hematogenous infection may take place from any primary focus, or through wounded oral mucosa. Lymphatic infection occurs most frequently through the tonsils. Infection around the third molars may infect the parotid glands and involve the salivary glands; because these lymphatics drain into the glands at the angle of the jaw following the retrograde stream. This happens in case of epithelioma of the tongue, or mandible, and would therefore be likely in tuberculosis as well.

Infection through the lymph-streams may occur from the external auditory canal, in case of a tuberculous ear, as occurred in de Paoli's first case. Considering the frequency of aural tuberculosis, it seems odd that the parotid is not more often affected. The lymphatics from the external two-thirds of the eyelids and from the upper part of the nose also pass from the parotid lymph-nodes, but neither are these parts often affected by tuberculosis. Furthermore, the posterior buccal lymph-nodes have vessels passing to the parotid nodes and penetrating the muscle in the region through which Steno's duct perforates.

Carmody believes that the canalicular infection is most likely. Pinoy's experiment (quoted by Püppel) would tend to show that infection by any of the three ways noted above is perfectly possible. He infected the parotid

gland experimentally by injection of bacilli into the parenchyma, as well as by introduction into the duct after paralysis of the secretary nerve.

SUMMARY

(1) Tuberculosis of the parotid gland is rare and is often mistaken for malignancy, such as syphilis or mixed tumor.

(2) Two distinct types are usually found: The first, chronic or fibroid type, which is incapsulated, and may not produce symptoms for months or even years; the second type is acute inflammatory, which is diffuse and runs its course in a few days or a week.

(3) Tubercle bacilli gain entrance into the parotid gland by one of three ways: Canalicular, hematogenous or lymphatic, with the first being the most common.

(4) Symptoms consist of swelling of the gland, either as a circumscribed or fluctuating tumor, or more diffuse with an occasional soft spot. The second is usually adherent, red, tense, shiny and œdematous. Pain is a late sign and the glands of the neck are not involved.

(5) Diagnosis is likely to be difficult unless it is confirmed by biopsy.

(6) Prognosis is good, as the affection is purely local.

(7) Treatment is purely operative, and even where facial paralysis takes place from injury to the facial nerve, either by disease or operative trauma, there is recovery in most instances.

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METASTATIC EPIDURAL ABSCESS OF THE SPINAL CORD

RECOVERY AFTER OPERATION

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Emergency operations for relief of compression of the spinal cord in cases other than traumatic origin are not common, according to the literature, and are performed usually for infections about the spinal canal. The reported high mortality and discouraging post-operative results probably can be traced to the virulence of the organism and the lowered resistance of the patient, but delayed or neglected operative interference may be a contributory factor.

The literature on this general topic was reviewed by Dandy[4] in 1926. In addition to the ten cases of epidural abscess of metastatic origin, which he cited and tabulated, we have been able to find reports of four cases; namely, those of Spiller, Braun, Bensheim,[1] and Pulvirenti (tabulation). For clarity and emphasis, in this paper, consideration has not been given to cases of epidural abscess which were not of metastatic origin, other than to remark that in one case of epidural abscess, due to direct extension of a suppurative process involving neighboring structures, operation was performed and the patient subsequently recovered.[8] Of the fourteen cases of metastatic abscess which have been reported, operation was performed in three. One of the patients died,[5] the outcome is not stated in the second case [3] and Pulvirenti's patient recovered from the operation and was walking without symptoms within a year. Thus, the case reported by Pulvirenti is the only case of metastatic epidural abscess we know of in which diagnosis was made sufficiently early to permit of successful treatment and recovery. We are reporting in detail a case which has been under our care for more than a year and a half.

Report of Case.—An emotional, large, and obese nurse, aged twenty-eight years, came to the clinic December 8, 1929, complaining of an infection of the nose which had begun that day and which was associated with swelling of the entire face. December 11 she complained of pain in the left ear; examination revealed otitis externa, which cleared up readily under treatment. On the same day nasal discharge was established and röntgenograms of the sinuses were cloudy; improvement set in gradually, and röntgenograms of the sinuses, made December 20, were negative. December 21 a pain suddenly developed in the left costovertebral angle, and was projected along the costal margin to the mid-axillary line. The pain gradually increased in severity, and by December 25 the patient was writhing in pain. She was admitted to hospital at that time as an emergency patient. The temperature was 99.4° F., and the pulse rate, 66 beats a minute. There was tenderness in the left costovertebral angle. Leucocytes numbered 13,500 in each cubic millimetre of

TABULATION

Summary of Four Cases of Metastatic Extradural Abscess of the Spinal Cord, Reports of Which Have Been Found Since Dandy's Review *

Case	Reporter and date	Age, years and sex	Primary infection	Situation of metastatic extradural infection	Symptoms of onset, distribution and type of pain	Paralysis	Bladder or rectum, sensation	Common signs of meningeal irritation	Leucocytes in each cm. of blood and temperature	Reflexes	Spinal fluid	Duration of life	Clinical diagnosis	Comment
1	Spiller 1921	36 M	Furuncle on neck	Sixth thoracic vertebra	Backache; lower thoracic and upper lumbar vertebræ and legs; flash-like, burning, increasing severity	Progressive paraplegia complete in one month	Retention with overflow incontinence; absent below distribution of seventh thoracic nerves	Spinal rigidity and tenderness; Kernig's sign positive	Not recorded	Absent	Yellow, clotted	Sixty days	Not recorded	Necropsy; leptomeningitis and right empyæma
2	Braun 1922	30 M	Injury left scapula followed by local abscess	Entire epidural space	Pain; present but not described	Absent	Not recorded; negative	Spinal rigidity and tenderness; Kernig's sign positive; headache	Leucocytes not recorded; temperature elevated	Negative	Negative appearance; few staphylococci on culture	More than three days	Meningitis	
3	Bensheim 1928	M	Generalized furunculosis (staphylococcus)	Posterior part of extradural space of lumbar and thoracic vertebræ	Pain; lumbar portion of spinal column and legs; severe and electric-shock-like	Paraplegia, flaccid, appeared overnight	Retention with overflow incontinence; absent below distribution of second sacral nerves; hyperæsthesia with gradual transformation to hypæsthesia	Spinal rigidity and tenderness; Lasègue's sign positive bilaterally; headache	6,600 on admission increasing to 10,000; temperature 38°C. (100.4°F.)	Patellars diminished; Achilles definite	Not known	Almost nine days	Septic myelitis	Necropsy; abscess along spinal column, right side; phlegmonous abscess of posterior aspect of left psoas muscle with extension of yellow pus along spinal nerves into spinal canal and down sciatic nerve to its emergence from the pelvis
4	Pulvirenti 1921	19 M	Furunculosis left arm (staphylococcus)	Third to fourth lumbar vertebræ	Lumbar pain, paraplegia, lumbar region	Paraplegia	Not recorded; diminished in legs	Not known	Not recorded; temperature slightly elevated at onset	Not recorded	Pus obtained between third and fourth lumbar vertebræ	Recovered	Acute purulent lumbar spondylitis	Operation; three months later, dismissed with slight spastic gait; eight months later, normal central nervous system

* Our case is not tabulated here, since the details are given in the text.

blood with 7 per cent. neutrophiles. The patient gradually grew worse and the temperature fluctuated from 99.4° F. to 103° F. with corresponding elevation of pulse. The pain became worse and relief was not obtained with opium.

Neurological examination, December 27, gave objectively negative results, except that on flexion of the head on the chest, even to a slight extent, there was marked aggravation of the costovertebral pain. There was a suggestion of Kernig's sign bilaterally, particularly on the left; also on the left, the manœuvre employed in elicitation of this sign was associated with exaggeration of the pain. The urine was negative to examination. The cerebrospinal fluid was clear and colorless; it was under normal pressure, and the pressure responded promptly to compression of the jugular veins. The Wassermann and Nonne tests of the cerebrospinal fluid gave negative results, and there were two small lymphocytes in each cubic millimetre. December 30 neurological examination was again objectively negative, except for aggravation of the pain on bending the head forward, and on Lasègue's manœuvre. There was marked tenderness over the spinous process of the twelfth thoracic vertebra and laterally along the course of the twelfth thoracic nerve. Cultures of the urine were negative. Röntgenograms of the thorax and spinal column were of normal appearance. December 31 the patient was unable to void and was catheterized after the bladder had risen halfway to the navel without inducing discomfort.

January 1, 1930, leucocytes numbered 15,600 in each cubic millimetre of blood. The patient complained of pain in both legs and there was involuntary defecation. January 2 she was exquisitely sensitive from head to foot. The neck was not stiff, there was no longer pain on forward flexion of the head on the chest, and there was no tenderness over the spinous process of the twelfth thoracic vertebra. Neurological examination was again objectively negative.

On the morning of January 4 the patient complained of weakness in both legs. By noon, partial flaccid paralysis of both legs had developed, and the tendon reflexes were obtained with difficulty. Also, slight diminution of common sensation over the legs, the posterior aspects of the thighs, and the buttocks was noted. Another spinal puncture, made in the space between the third and fourth lumbar vertebræ, at 1 P.M., under ethylene anæsthesia, revealed clear, lemon-colored, viscid fluid under pressure of 170 millimetres of water, which became opalescent and coagulated on standing. There was no response on compression of the jugular veins. At 2.30 P.M. neurological examination revealed flaccid paralysis and areflexia of both legs. (Fig. 1.) Loss of sense of position in both big toes and of vibratory sensation over the malleoli was noted. There was abolition of sensation of touch, pain, and temperature over the feet and legs, the posterior aspect of both thighs, and the buttocks, and there was partial loss of the latter qualities over the anterior aspect of the thighs. The level at which sensory disturbance began approximately coincided with the inguinal ligaments.

In view of the inflammatory course of the condition, and the increasing leucocyte count, the paraplegia and spinal subarachnoid block, with Froin's syndrome, and the situation and aggravation of the sensory disturbances following lumbar puncture, a diagnosis was made of extradural abscess, and immediate exploration was advised.

At 4.50 P.M., January 2, the spines and laminæ of the eleventh and twelfth thoracic and first and second lumbar vertebræ were removed. An epidural abscess ruptured during removal of the bone at the level of the twelfth thoracic vertebra. (Fig. 2.) Thick, creamy pus escaped. After the incision had been carried up to the level of the eleventh thoracic vertebra and down to the second lumbar vertebra, the abscess was found to be fairly well walled off. Reddish granulation tissue, which composed the walls of the abscess, could be easily stripped off the dura. The dura pulsated normally after evacuation of the abscess and removal of its walls.

The entire wound was swabbed with tincture of iodine; Penrose and iodoform drains were inserted, and closure was effected with sutures of silkworm-gut. On account of impaired function of the vesical sphincters, a retention catheter was inserted while the patient was on the table. The entire operation was carried out under ethylene anæsthesia.

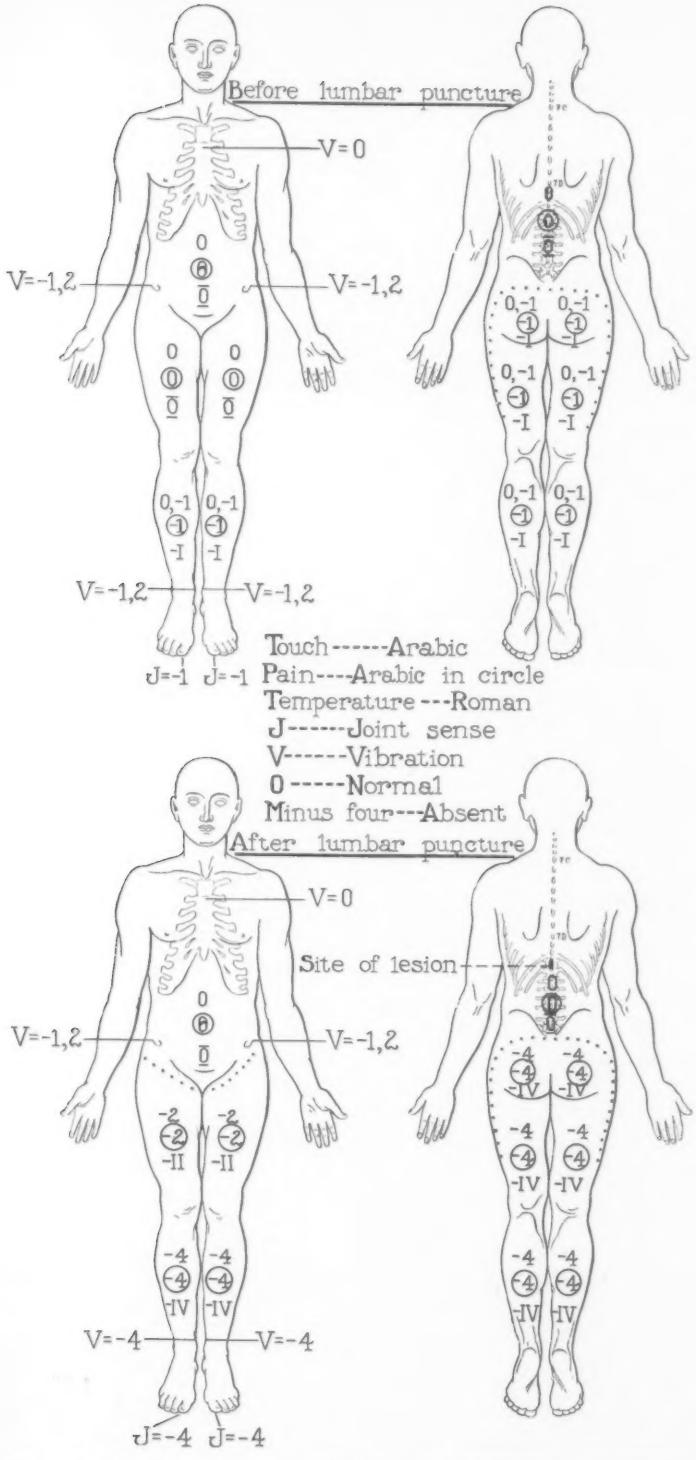

FIG. 1.—The marked sensory change which occurred following lumbar puncture, localizing the lesion.

Cultures of the pus and inflammatory tissue removed at operation, made in glucose brain broth and blood agar, revealed staphylococcus.

By the morning of January 5 the patient was almost completely relieved of pain, and there was no change in power, sensation or control of sphincters. Blood cultures revealed one to two colonies of staphylococci on blood agar at the end of forty-eight hours. Two days later the patient described the sensation of tingling in both legs and was practically without pain.

January 9 blood cultures were again positive for staphylococcus. Coarse clonic movements of both thighs were first observed January 13. A positive Babinski's sign had appeared two days before.

January 31 the patient complained of feeling very tired and of severe pain on involuntary activity of the legs. Examination revealed paresis of the lower half of the anterior abdominal wall, associated with partial anæsthesia which extended as high as the umbilicus. Although the wound was draining profusely, signs of returning compression necessitated removal of the sutures, to allow even more free drainage and daily irrigations. Following this procedure, there was immediate improvement in the patient's condition, and February 13 she complained for the first time of distention of the urinary bladder when the catheter

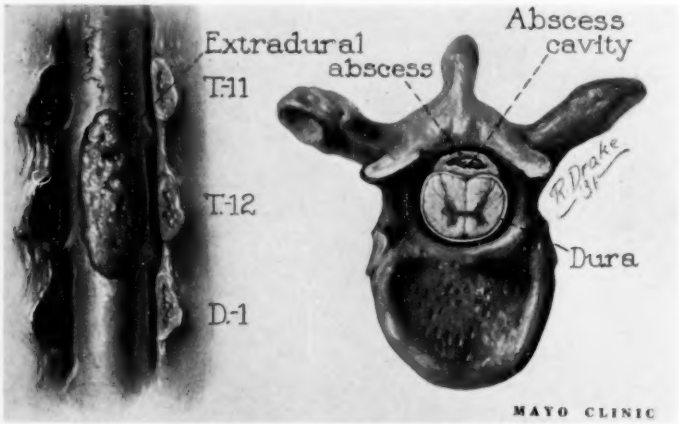

FIG. 2.—Situation and size of abscess.

was clamped. At this time there was noted, also, slight return of voluntary control of toes and feet and daily physical therapy was begun.

Following an interval of six days, during which the temperature had been normal, it suddenly rose to 104° F., and a red, indurated, tender, subcutaneous mass was discovered over the trochanter of the right femur. This was incised and drained. The bacteriological report revealed that the pus was of staphylococcic origin. By March 3 the retention catheter could be removed, and the patient began to void voluntarily.

March 15 the patient complained of pain in the region of the left shoulder and again the temperature was elevated. A subcutaneous abscess on the left shoulder was evacuated, the pus containing staphylococcus. After a short interval the wounds healed satisfactorily, with the exception of the laminectomy wound, which slowly granulated and healed over.

By April 25 the patient was able to sit up in a chair, and the positive neurological evidence consisted of considerable loss of power in the muscles of the legs; there was also moderate weakness of the muscles, the tendons of which bound the popliteal space. The patellar and Achilles reflexes were active, and Babinski's sign was present bilaterally. Joint and vibratory sensations were absent in the big toes and at the malleoli, respectively. Slight perception of touch, pain, and temperature indicated return of sensation on both sides, below the level of the umbilicus, and the patient was able to be up and about with assistance or in the orthopædic "walker." So much had she improved that by May 21

she was able to walk 100 feet in the hospital corridor. She continued in the hospital until July 14 with daily physical therapy and exercise in the walker. The wounds were healed and the sphincters essentially were competent. She was transferred to a convalescent home to continue with physical therapy and exercise, and by July 26, assisted by two nurses, she was able to walk without the walker.

The patient continued to improve, and when examined October 22 there was found to be slight to moderately marked spastic weakness of the muscles of the gastrocnemius group associated with active tendon responses and Babinski's and Chaddock's signs bilaterally. The sense of position had not returned in the big toes and was slightly diminished in the ankles. Vibration was not perceived over the malleoli and only feebly over the iliac crests. Below a line drawn through the umbilicus, common sensation was diminished in acuity. The patient was able to walk with a moderately ataxic, somewhat spastic gait, assisted by one attendant.

When the patient was next seen, in November, she could walk without any assistance, and was gradually but progressively improving. By January 26, 1931, she was able to climb stairs holding to the hand rail. March 3 objectively she had shown little improvement since her examination on October 22, 1930, but she was walking better and was able to take 600 steps on the level and to climb up and down twenty-two steps four or five times daily. She returned for observation July 13, 1931, and was found to be markedly improved. Although she was still receiving physical therapy, she was able to walk a considerable distance. Neurological examination gave evidence of slight weakness of the right peroneal muscles and of the plantar flexors of the left foot. There was moderate weakness of the left peroneal muscles and of the dorsiflexors of the big toe. In addition, the deep reflexes of both legs were moderately exaggerated and were associated with sustained ankle clonus and positive Babinski's sign. Common sensation and deep sensation were essentially the same as on October 22, 1930. Gait was somewhat ataxic and spastic. The patient weighed 223 pounds, and she was referred to the diet kitchen with the suggestion that she reduce fifty pounds; she was advised also to increase her physical activities. August 25, 1931, she weighed 211 pounds and was improving in strength.

Comment.—There is no clear-cut syndrome characteristic of epidural abscess. In general, the clinical picture is likely to be characterized by evidence of an acute inflammatory process associated with symptoms of moderately rapid progressive spinal compression. Pain may be a prominent symptom, sudden in onset, and rapidly increasing in severity; in six of the fourteen cases recorded in the literature, and in our case, the pain had these characteristics. The pain may be radicular in distribution, as it was in our case. Tenderness over the site of pain was present in six instances in the literature and was very marked in our case.

The development of paraplegia in only eight of the reported cases suggested that the patients were overwhelmed by the infective process before sufficient spinal compression had occurred to render them paralytic; in our case, and that of Bensheim, the paraplegia was flaccid. In eight cases in the literature, the status of sensation is recorded; in four cases, sensation was lost at and below the level of distribution of the affected part of the spinal cord; in two cases, sensation was diminished; in one case, it was said to have been negative, and in the remaining two cases, there was hyperæsthesia.

In the beginning of our patient's illness, she was hyperæsthetic. At noon, on the day of operation, examination revealed only slight diminution in the acuity of perception of cutaneous sensation, but following lumbar puncture

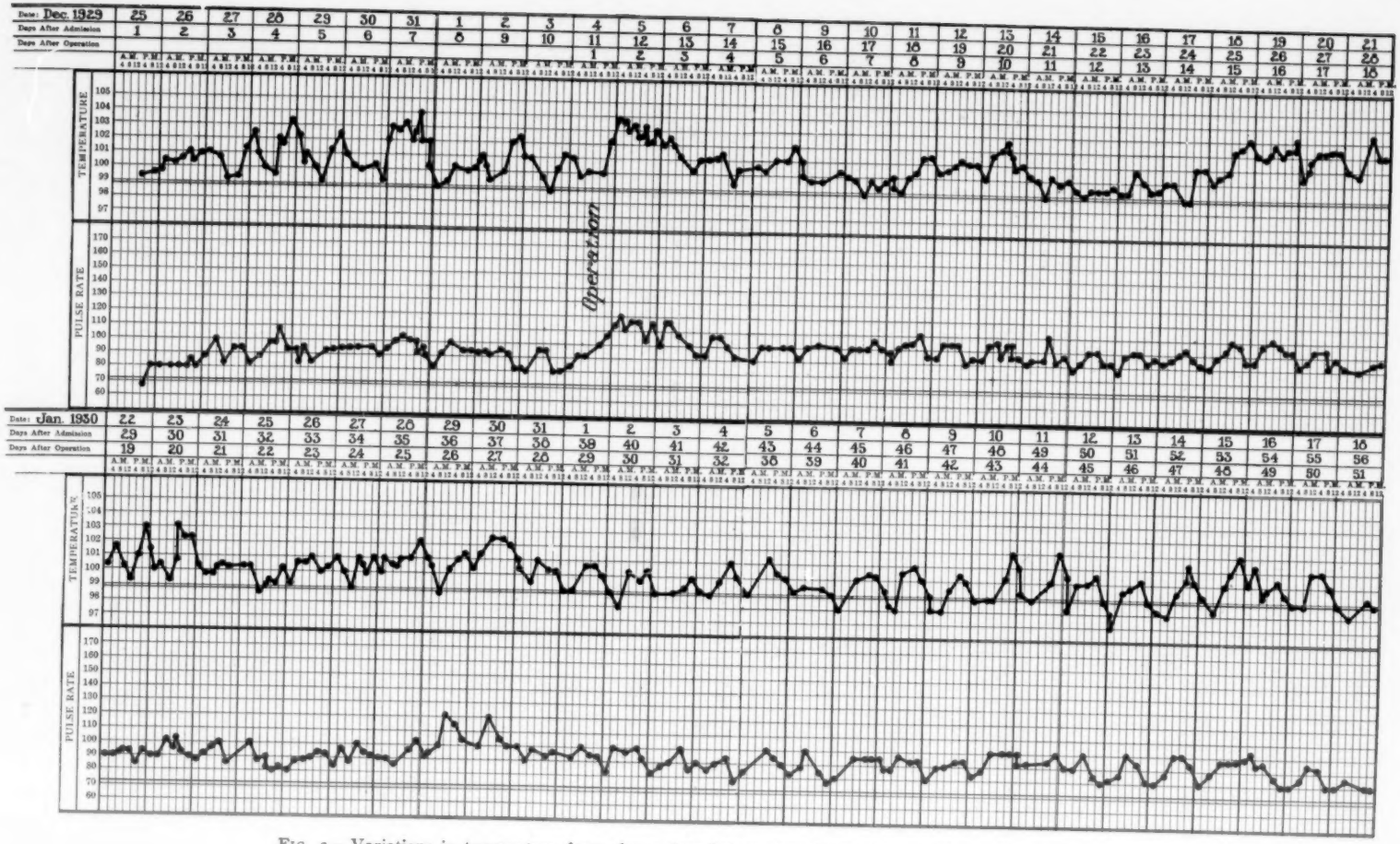

FIG. 3.—Variations in temperature from eleven days before operation to fifty-one days after operation.

and withdrawal of cerebrospinal fluid, sensation was lost below the level of distribution of the third lumbar segment of the spinal cord and obtunded over the distribution of the first three lumbar segments.

Urinary retention developed in our case, and in four of the fourteen cases of which histories are recorded in the literature. Kernig's and Lasègue's signs were reported as being positive in four instances in the literature, but in our case these were not only present but were more pronounced on the side of the pain. The leucocyte count is recorded in only three cases in the literature, and ranged from 6,600 to 18,000, whereas, in our case, it was first recorded as 13,500 and subsequently fell to 6,500, only to rise to 19,000 at the time of operation and to 23,000 forty-eight hours thereafter. Elevation of temperature was recorded in seven cases and ranged between 38° and 41° C., and was manifest in our case, with extremes varying from 99.4° to 104°F. (Fig. 3), which is approximately equivalent to 37.5° to 40°C. The cerebrospinal fluid was examined in five cases, and in two cases, diplococci were found. In one case[2] a few colonies of staphylococci were observed after culture. In one case,[7] the fluid was yellow and clotted, similar to ours. In Pulvirenti's case, pus was obtained by spinal puncture. Nine days before operation, the cerebrospinal fluid of our patient was entirely normal, but at 1 P.M. on the day of operation lumbar puncture revealed clear, lemon-colored, viscid fluid which coagulated on standing; there was no response of the fluid in the manometer on compression of the jugular veins.

In three instances in the literature, the infective process involved the greater part of the entire epidural space; in six others, and in our case, the lesion was in the thoracic or thoracolumbar regions, and in one case the third and fourth lumbar segments were involved. The duration of life of the patients in ten of the fourteen cases reported in the literature is recorded. Braun's patient lived more than three days, Bensheim's patient almost nine days, and Spiller's patient lived sixty days. Pulvirenti's patient recovered.

The symptoms group themselves into two categories. There are those to be associated with inflammatory disease, such as headache, general malaise, fever, and leucocytosis, with or without evidence of bacterial invasion of the cerebrospinal fluid. In the entire group of cases, there is evidence of infection at some site more or less distant from the spinal column. In the second group of symptoms are those suggestive of irritation of one or more spinal nerve roots. The outstanding symptom in this group is pain, which is often of sudden onset and increasing severity, and which may manifest the characteristics of a so-called root pain. This may be followed by such evidence of spinal compression as weakness, diminished sensation, and failure of function of the sphincters.

SUMMARY

In a review of fourteen cases of metastatic epidural abscess of which the histories are recorded in the literature, only one was discovered in which the

patient had recovered. An additional case is reported, in which the condition was diagnosed, the patient operated on, and recovery took place.

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PULMONARY EMBOLISM AND INFARCTION

ANALYSIS OF SIXTY-FOUR VERIFIED CASES

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This study is based on sixty-four cases of pulmonary embolism with or without infarction coming to necropsy in the Albany Hospital from 1921 to 1929 inclusive. The incidence of this much-feared complication varies in different statistics. Petrén[1] comments on the increased frequency of these cases during the last twenty-five years. Killian[2] noted that the percentage incidence of all cases of fatal pulmonary embolism gradually increased from 0.085 per cent. in 1919 to 0.3 per cent. in 1920. The primary site of the thrombus may occur anywhere in the venous circulation. It may even be primary in the lung itself (autochthonous). Frothingham[3] reported a very interesting case of autochthonous thrombosis, resulting from acute lesions in the arteries, where the thrombosis began in the smallest branches of the pulmonary arteries and propagated centripetally toward the larger branches, producing multiple small infarctions of all ages.

As stated by Aschoff,[4] thrombosis is a function of a number of variables. Much has been written on the importance of the various factors which enter into the formation of a thrombus. The exact mechanism producing more or less immediate death when only a small portion of the pulmonary circulation is obstructed by an embolus is still unknown. It appears that the circulatory cessation precedes the respiratory. Mann[5] has been able to produce death experimentally only by a more or less complete blocking of the pulmonary circulation. Haggart and Walker[6] experimentally showed that total pulmonary occlusion sets up a severe and immediate reaction, as evidenced by a rapid and marked dilatation of the heart. The minute volume output of the heart becomes less. The pulmonary pressure rises sharply, then gradually falls toward zero; whereas the systemic blood-pressure begins to fall immediately and does not recover, while the respirations become irregular and shortly afterwards cease altogether. In the production of multiple embolism by injecting large amounts of potato starch granules into the jugular vein of goats, Dunn[7] showed that the venous pressure rises while the arterial falls, followed by death. In cats[6] from 52 to 66 per cent. and in dogs[8] 75 per cent. of the pulmonary circulation can be shut off without producing significant variations in the general circulation. When one pulmonary artery is occluded by ligation, the pulmonary blood-pressure rises but there is no effect on the carotid pressure and on the rate, output and size of the heart —Underhill,[9] Haggart and Walker, Welch,[10] Plumier,[11] Gerhardt.[12] Experimentally Schlaepfer[13] found that the lung on the intact, non-ligated side

underwent a compensatory mechanism—chronic dilatation of the arterioles and capillaries and dilatation of all alveoli, but no fibrosis. The lung on the ligated side showed marked stasis of blood, diapedesis, and finally fibrosis. Kawamura[14] had already long ago noted this extreme connective-tissue proliferation of the lung on the ligated side and its subsequent marked contraction.

At the present time, more and more interest is being paid to the operative treatment of embolism of the lungs by the Trendelenburg operation as more and more successfully treated cases are being reported—Meyer,[15] Matas,[16] and Nystrom.[17] Even after the successful performance of Trendelenburg's operation, the risk of new emboli is very great, and Westerborn[18] warns us that this should be kept constantly in mind in the post-operative treatment of the patient and the estimation of the prognosis.

Incidence.—From 1921 to 1929 inclusive, sixty-four cases of pulmonary embolism came to necropsy in the Albany Hospital. These were divided as follows—post-operative, twenty-five cases; post-traumatic, three cases; medical, thirty-six cases. During this same interval, there were performed 810 necropsies which gives a necropsy incidence for embolism of 7.9 per cent. There was a total of 3,031 deaths in the hospital or a mortality incidence for embolism of 2.1 per cent. There was admitted to the hospital a total of 62,935 patients. The morbidity from pulmonary embolism will then be 0.102 per cent., or about one per 1000 hospital population. From Table I it is seen that the percentage autopsy incidence of embolic cases varies from a low 0.65 per cent. to a high 14.52 per cent.

TABLE I

Incidence of Embolism in Necropsied Cases

Author	Year	Number of necropsies	Number of embolic cases	Percentage of embolic cases
Rupp	1921	12,971	657	5.07
Hedinger and Christ	1922	3,000	436	14.52
Naegeli	1925	4,916	43	0.87
McCartney	1927	9,275	73	0.79
Stöhr and Kazda	1928	20,654	134	0.65
		714	13	1.82
		2,664	29	1.08
Farr and Spiegel	1929	1,116	30	2.68
Gruber	1930	15,867	271	1.71
Hosoi	1931	810	64	7.90

There were thirty-five males to twenty-seven females or 1.4 : 1 for embolism. The ratios for total admissions were 0.9 : 1; for total deaths, 1.5 : 1; and for necropsies, 1.6 : 1. This means that more males die and are autopsied in this hospital. Hence, sex has probably no significance in the etiology of embolism.

Among the men, 61 per cent. of the cases occurred between fifty-one and seventy and among the women, 73.3 per cent. between forty-one and fifty

years of age. As shown in Chart 1, the age incidence of embolic cases suddenly increases from forty years on for males with the mode at fifty-one to sixty years, and from thirty on for females with the mode at forty-one to fifty years. Similarly, the highest age incidence in Axhausen's[19] cases was in the sixth decade. Zurhelle[20] found the majority of his gynecological cases with embolism lay between thirty-six and fifty-five years with the mode at forty-one to forty-five years. In de Quervain's[21] cases, the highest incidence was between fifty and sixty-nine for both male and female. About two-thirds of all cases of both Killian[2] and Gruber[22] occurred after fifty years of age. On the other hand, Hampton and Wharton[23] found only one of their fifty-one embolic gynecological cases above fifty years of age and 66 per cent. between twenty and forty years.

As regards the seasonal influence, no reliable conclusions can be drawn

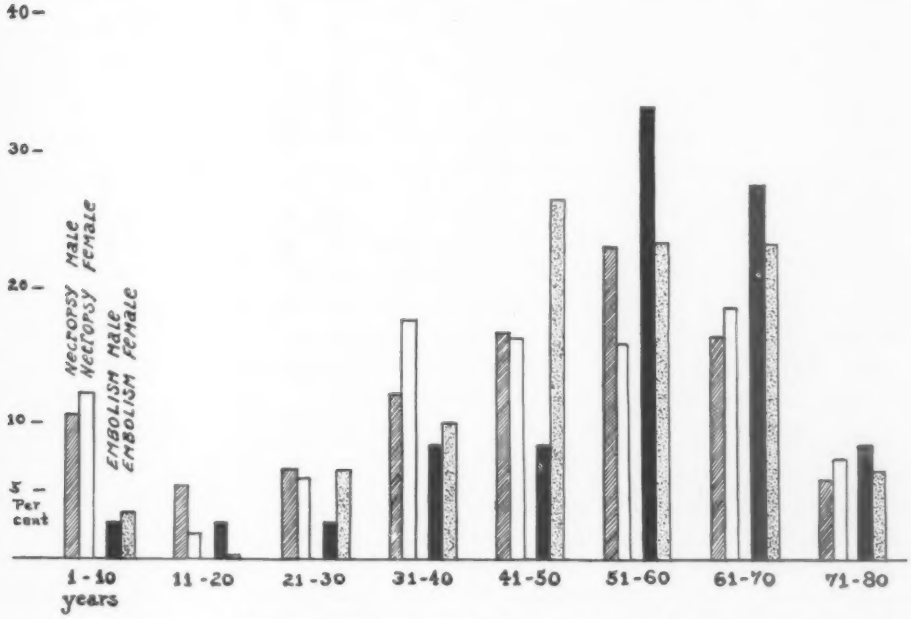

CHART 1.—Histogram of percentage frequency of necropsies and pulmonary embolism in each decade for male and female.

from this study. De Quervain found the highest incidence in February and March, but the percentage figures were generally so irregular that nothing definite was obtained. On the other hand, in Geissendörfer's[24] cases, the highest incidence was in May and the lowest in March. The highest percentage in the Albany series occurred in mid-winter and mid-summer, but we find that the greatest percentage of necropsies also occurred in the summer and winter months. This shows that, as Günther,[25] and Hosoi and Alvarez[26] have pointed out, every percentage figure should be corrected by a factor dependent upon the distribution of the group from which the cases studied were obtained.

Post-operative cases of embolism.—Any study of post-operative embolism

must necessarily take into consideration the incidence of thrombophlebitis which may or may not lead to embolic phenomena. There were forty-seven cases or 0.16 per cent. of post-operative phlebitis including eight cases of pulmonary infarction, forty-four of which improved and three died. These three did not come to necropsy, but one gave typical symptoms of cerebral embolism and one, pulmonary infarction. Twenty-four of these cases followed general surgical operations and twenty followed gynecological operations. A large majority of these had thrombophlebitis of the femoral veins. Cordier[27] from a study of 232 collected cases concluded that phlebitis occurs in about 2 per cent. of all abdominal operations.

TABLE II

Incidence of Femoral Thrombophlebitis Among Post-operative Cases

	Right	Left	Both sides	Not stated	Total
General surgery	4	15	1	1	21
Gynecology	8	10	2	—	20
	12 (29.3%)	25 (61.0%)	3 (7.3%)	1 (2.4%)	41

Table II shows that 61 per cent. of the post-operative femoral thrombophlebitis occurred on the left side. Sixty-four per cent. of the eighty-seven cases of phlebitis reported by Brown[28] occurred in the left leg. In Hampton and Wharton's 205 cases, the vessels of the left lower extremity were involved in 66 per cent. Cordier found that in about 92 per cent. of 232 collected cases the phlebitis occurred in the left saphenous or femoral veins. Various theories have been brought forth to explain this left-sided preponderance. A slowing of the venous stream on the left side has been stressed by many, especially when left-sided femoral thrombophlebitis occurs after right-sided abdominal operations. This slowing is believed to be caused by the greater length and obliquity of the left iliac vein, by pressure of the distended recto-sigmoid on the iliac veins, and by pressure of the right common iliac artery. McMurrich[29] found that valves or adhesions within the common iliac veins were much more frequent on the left side than on the right, dividing the lumen of the vein into two passages. Stasis and slowing of the blood-stream have been proved experimentally that *per se* they cannot produce thrombosis. The blood in the artery or vein included between two sterile ligatures was found fluid for hours, days and weeks by Hunter,[30] Durante,[31] Glénard,[32] Baumgarten,[33] Raab,[34] Senftleben,[35] and Miller and Rogers.[36]

Among the autopsied cases of post-operative thrombophlebitis, there were only three cases in which thrombosis of the iliac and femoral veins was demonstrated. All three cases were non-gynecological. In two, the left common iliac vein was involved, and in one the right iliac with peripheral extension. In their studies on experimental thrombophlebitis and lymphatic obstruction of the lower limb in dogs, Homans and Zollinger[37] concluded that the basic lesion is always in the common or external iliac vein, however far the thrombophlebitis may extend peripherally.

Clark[38] found that femoral thrombophlebitis occurs with startling regularity after the eighth day, appearing most frequently about the fifteenth day after operation. Schenck's[39] cases occurred from the sixth to the twenty-second day post-operative. In the cases here presented, the onset post-operatively ranged from the seventh to the twenty-eighth day, 50 per cent. occurring between the eleventh and fifteenth days.

According to Mahler,[40] the diagnosis of thrombosis can be made with certainty only by a study of both the pulse and temperature curves. He particularly stressed the step-like increase of the pulse, associated with a normal temperature, which takes place several days before the thrombosis becomes evident. Payr[41] often saw isolated rises of pulse or temperature but concluded that they were of doubtful value. Hampton and Wharton found that thrombosis of a large peripheral vein may be present without any local signs or symptoms whatever. They, however, observed in almost all their cases a low protracted febrile course, 99 to 100° F. in the convalescence preceeding the infarction and this they attributed to the presence of a thrombus. Glynn[42] stated that

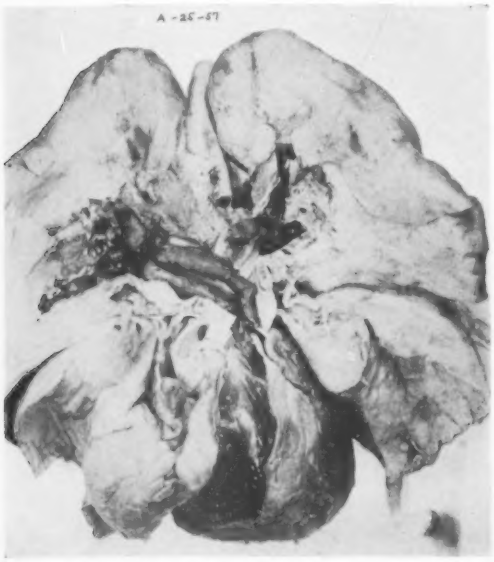

FIG. 1.

FIG. 2.

FIG. 1.—Photograph of heart and lungs with the pulmonary artery opened. An embolus is coiled up in typical fashion in the right and left pulmonary arteries.

FIG. 2.—Photograph of emboli removed from the main pulmonary artery and the right ventricle and reconstructed as closely as possible to the probable original form. This composite picture gives an idea of the size, extent, and branching of the original thrombus which may have started in the iliac vein and its tributaries.

the development of primary pulmonary thrombosis was often suggested by the slight rise in pulse rate without corresponding rise in temperature. In the Albany series, where there was no infection of the operative field and where the respiration was normal and the temperature between 98 and 99, the pulse ranged between 80 and 116. In those clean operative cases where the temperature reached 100 or even 103 temporarily, the associated pulse rate was as high as 140 and the respiration rose to between 30 and 40. The discrepancy noted above between temperature and the pulse became noticeable in some of these cases when the temperature fell down to within normal limits. This discrepancy is certainly suggestive but only relative, since there were cases where the pulse remained at normal levels.

It has not always been possible to determine at necropsy the location of the thrombus formation which gave rise to the pulmonary embolism. Often

the only clue that the thrombus may have originated in the femoral, iliac or other large veins is the large size of the embolus or its coiled-up appearance (Fig. 1), the branching structure of which may be demonstrated by floating the embolus in water. Sometimes, by reconstruction of the embolus, it has been possible to surmise that a large vein was the source of the embolus (Fig. 2).

Table III

Incidence of Post-operative Embolism

Author	Year	Number of operations	Embolic cases No.	Embolic cases %	Type of Operation
Cutler and Morton	1917	3,490	6	0.17	General surgery
Hampton and Wharton	1920	21,000	51	0.24	Gynecological only
Eisenreich	1920	3,981	12	0.30	Gynecological only
Rupp	1921	22,689	51	0.26	General surgery
		2,769	10	0.36	Gynecological only
Cutler and Hunt	1922	1,604	2	0.12	General surgery
de Quervain	1925	56,020	249	0.46	General surgery
Naegeli	1925	15,343	21	0.67	General surgery
Farr and Spiegel	1929	12,813	22	0.171	General surgery (blunt dissection)
		12,615	21	0.167	General surgery (fine dissection)
Fuller	1930	1,478	10	0.67	General surgery
Killian	1930	22,413	44	0.19	General surgery
Geissendörfer	1930	20,960	46	0.22	General surgery
Hosoi	1931	22,240	27	0.12	General surgery
		6,265	6	0.10	Gynecological only

Incidence of post-operative embolism.—As shown in Table III, the percentage incidence of post-operative embolism varies approximately from 0.12 to 0.67 among the general surgical patients and from 0.10 to 0.36 after gynecological operations. Cutler and Morton[43] observed that one in every 581 operations is liable to be followed by pulmonary embolism. Ochsner and Schneider[44] found only one death from pulmonary embolism in every 2,385 operations. The table shows that the incidence of embolism in the Albany Hospital is among the lowest when compared with that of other institutions. Between the years 1921 and 1929 inclusive, there were performed 22,240 general surgical operations and 6,265 gynecological operations. This gives a post-operative incidence for embolism of 0.12 per cent. for general surgical patients and 0.10 per cent. for gynecological patients, or a total of less than 0.12 per cent. for the whole hospital. Contrary to what has been found in other clinics, embolism occurs less frequently among the operated gynecological patients as among those admitted to general surgery in this hospital. From a personal experience of 1000 consecutive cases, Dannreuther[45] found an incidence of 0.4 per cent. for thrombophlebitis and 0.3 per cent., for embolism after pelvic surgery. Rupp[46] gave an incidence of thrombophlebitis of 0.3 per cent. among 2,769 operations on the female genital tract; Morley,[47] 0.62 per cent. among 1,756 gynecological operations; Burnham,[48] 0.81 per cent.

among 11,655 operations; and Hampton and Wharton, about 1 per cent. among 21,000 gynecological operations. Schenck[49] found that among 49,161 gynecological operations reported by twelve writers, there were 566 instances of thrombosis or 1.15 per cent.

It is generally believed that operations on the myomatous uterus are notorious for their thrombotic and embolic complications. Of the twenty cases of post-operative thrombophlebitis, nine or 45 per cent. had a myomatous uterus. Of the five necropsied gynecological cases, only one had multiple myomas. Among Cordier's 232 cases, phlebitis followed hysterectomy for fibroids in 30 per cent. of the cases. Thirty-three and seven-tenths per cent. of Hampton and Wharton's 205 cases were operation on for myomas of the uterus. However, von Lichtenberg[50] found only 0.8 per cent. with pulmonary complications and 0.7 per cent. with embolic phenomena among the 1,479 operations for myomectomy. Also Schenck[39, 49] found that following 3,204 myoma operations reported by eight writers, there was thrombosis in only 3 per cent. Yet, 39.6 per cent. of his own forty-eight cases of phlebitis followed hysteromyomectomy and myomectomy. There were only two cases (0.4 per cent.) of embolism following 451 myoma operations at the Münchener Frauenklinik.[51] In Klein's[52] series of 730 myoma operations, thrombosis occurred in 3.3 per cent. Among 654 operations for myoma, Zurhelle found eighteen (2.75 per cent.) cases of thrombosis and ten (1.5 per cent.) of pulmonary embolism. Thus, it is seen that in the larger statistics, the incidence of thrombosis after operations on the myomatous uterus averages about 3 per cent., which is not a high figure.

Table IV shows the types of operations which were followed by pulmonary embolism with or without infarction among the twenty-five cases. Twenty were performed under general ether anæsthesia, three under local, and two under spinal. Three were entirely non-abdominal—two radical breast amputations and one resection of the knee. There were five operations in the upper abdomen and seventeen (68 per cent.) in the lower abdomen. This is in agreement with the general belief that lower abdominal operations are more likely to be followed by thrombotic and embolic phenomena than those on the upper abdomen. In Henderson's[53] statistics, 44 per cent. of the abdominal operations were performed in the upper portion and 51.6 per cent. in the lower. Of the forty-three fatal pulmonary embolism cases of Naegeli,[54] 19 per cent. had operations in the upper abdomen, whereas in 51 per cent. the operations were gynecological, or in the lower abdomen. Lister's[55] statistics suggest that once an incision is made through the anterior abdominal wall, the liability to embolism depends on the age of the patient and not on the actual operation performed. Fuller[56] states that the length of the operation does not seem to bear any intimate relation to the occurrence of post-operative pulmonary complications.

Trauma.—The amount of trauma, which may occur depending upon the type of the operation and upon the surgeon, has been stressed by many as being of etiologic importance in thrombosis and embolism. Clark[38] believed

TABLE IV

Necropsied Cases of Post-operative Pulmonary Embolism

No.	Sex	Age	Operations	Infection at operative field	Days post-operative	Source of embolus—veins	Pulmonary infarction—lobes
1	F	79	Colostomy	Post-operative peritonitis	5	Undetermined	Left lower lobe
2	M	70	Suprapubic cystostomy	Clean	9	Vesical veins	None
3	F	68	Radical breast amputation	Clean	32	Bilateral femoral	Right lower and upper
4	M	74	Inguinal hernoitomy	Clean	9	Undetermined	Right and left lower
5	M	26	Appendectomy for ruptured gangrenous appendix	Local peritonitis	8	Undetermined	None
6	F	54	Panhysterectomy, bilateral salpingo-oöphorectomy, appendectomy	Clean	11	Small pelvic	None
7	F	6½	Appendectomy for ruptured appendix	Peritonitis with walled-off abscesses	3	Thrombi in many subperitoneal veins	None
8	F	52	Appendectomy	Clean	2	Undetermined	Lower
9	F	43	Panhysterectomy, bilateral salpingo-oöphorectomy	Clean	10	Undetermined	None
10	F	45	Supravaginal hysterectomy, bilateral salpingo-oöphorectomy, perineorrhaphy, appendectomy	Clean	7	Undetermined	None
11	M	72	Suprapubic prostatectomy	Acute cystitis	42	Undetermined	Right lower
12	M	35	Appendectomy	Clean	8	Prostatic plexus, external and internal iliacs	Right lower
13	M	61	Cholecystotomy followed by biliary fistula	Post-operative peritonitis with abscesses	?	Undetermined	Right lower
14	M	76	Gastroenterostomy	Clean	2	Right femoral	Left lower
15	F	57	Radical breast amputation	Clean	24	Iliac veins	Left upper and lower Right lower
16	M	60	Suprapubic cystostomy	Clean	31	Undetermined	Right lung
17	M	56	Colostomy	Clean	2	Undetermined	None
18	M	58	Mickulicz operation	Clean	10	Pelvic veins	Left lung
19	F	66	Cholecystotomy, bilateral salpingo-oöphorectomy, appendectomy	Clean	24	Left femoral	Left lower
20	F	61	Panhysterectomy, bilateral salpingo-oöphorectomy, umbilical herniotomy, appendectomy	Post-operative peritonitis	6	Undetermined	Lower
21	M	53	Appendectomy for gangrenous appendix	Local peritonitis	4	Undetermined	None
22	F	67	Cholecystotomy with drainage, ventral herniotomy	Clean	17	Undetermined	Righ lower
23	F	59	Ventral herniotomy, with lipectomy	Clean	15	Epigastric and iliac	None
24	M	61	Marsupialization for pancreatic cyst	Clean	20	Undetermined	None
25	M	40	Resection of knee	Clean	12	Undetermined	Left lower

that trauma caused by heavy retraction of the abdominal wall with retractors starts up a propagating thrombotic process in the deep epigastric veins. Yet, as shown in Table III, Farr and Spiegel[57] could find no difference in the incidence of pulmonary embolism after operations performed under blunt dissection or under fine dissection. McLean's[58] experimental work on etherized dogs is most interesting. He found that the crushing of a vein will not cause a clot at the point of crushing. The procedure can be repeated in forty-eight hours and still a clot will not form at the site. He concluded that endothelial damage is not *per se* a cause of thrombosis, thus confirming the work of Glénard[32] on traumatized arteries.

Infection.—Infection has been given much prominence in the causation of thrombo-embolic phenomena. From Table IV, it is seen that seventeen (68 per cent.) of the twenty-five cases can be regarded as being clean cases. In the remainder, the operative field was found grossly infected or became so post-operatively. In one case, the operative field was clean but a pyelonephritis was present. From a study of the necropsy material of 20,654 cases, Stöhr and Kazda[59] concluded that infection appears to play a secondary rôle in the causation of local thrombosis and that infection has no noteworthy significance in the origin of distant thrombosis. In 1887, Weigert[60] frequently found masses of micrococci inside marantic thrombi. In Welch's laboratory, Harris and Longcope[61] (1900) examined bacteriologically forty-four thrombi and demonstrated the presence of bacteria in thirty-four of these. Rosenow[62] in 1927 isolated the diplo-streptococcus from the embolus in each of the five necropsied cases of post-operative pulmonary embolism, following thrombosis of the iliac or femoral veins. There is no doubt that infection can be an important factor in the causation of post-operative thrombosis, but it does not explain those thromboses frequently occurring in cases without manifest infection. In none of the sixty-five cases of Möller[63] was it possible to demonstrate bacteria in the emboli. Indeed, McLean[58] found that crushing a vein with the subsequent introduction of a twenty-four-hour bouillon culture of staphylococci and again crushing the vein to grind the staphylococci into the walls of the vein will not produce a clot or thrombus at the site of the crushing. But he found that clots were formed if he introduced an infected thread into the vein or artery and allowed the thread to remain suspended in the lumen of the vessel, whereas no clots were formed if he repeated the experiment with sterile threads. He believed that infection and necrosis, or the toxins derived from an infectious and necrotic process, are probably the most important factors in the production of a thrombus.

Localization of embolus.—In the cases here reported, 42 per cent. of the post-operative emboli lodged in the lower lobes, more often on the right in the ratio of almost 2 : 1; 42 per cent. in the pulmonary artery or in one of its two main branches, the right being favored; 4 per cent. were multiple. The upper and middle lobes were not affected in these post-operative cases except in those with multiple embolism. Möller's[63] ratio of right to left lung

involvement was 5 : 3. In Tiedemann's,[64] Rupp's,[65] Hedinger and Christ's,[66] and de Quervain's[21] statistics, there was a marked predilection for the lower lobes, especially the right lower lobe. Welch[67] in 1899 wrote: "The course followed by an embolus in its travels is determined by purely mechanical factors of which the most important are the size, form, and weight of the plugs; the direction, volume, and energy of the carrying blood-stream; the size of branches and the angles at which they are given off; and the position of the body and its members. In accordance with these principles, we find emboli in the lower lobes of the lungs oftener than in the upper; and in the right lung oftener than in the left, the right pulmonary artery being larger than the left." Martin[68] ingeniously produced a thrombus in the femoral veins of dogs by injecting a mixture of saline, liquor ferri sesquichloridi and barium sulphate, loosened the thrombus thus formed, and watched it travel to the lungs under the Röntgen-screen. The thrombus at first traveled slowly through the inferior vena cava, but much more rapidly after it had passed the level of the diaphragm; then it was churned up in the right ventricle and rushed onward into the pulmonary artery and its branches, particularly in the lower lobes. The upper lobes remained relatively free.

TABLE V

Onset of Post-operative Pulmonary Embolic Symptoms

Weeks	1	2	3	4	5	6	Undetermined
Per cent.	32	32	16	8	4	4	4

Days post-operative.—Table V shows the number of post-operative days elapsing before the pulmonary embolic symptoms were first noticed. In three non-infected cases, the pulmonary embolism occurred on the second day. Sixty-four per cent. of the cases occurred in the first and second weeks post-operatively. One case each occurred in the fifth and sixth weeks. Examination of the autopsied cases of pulmonary embolism reported by Farr and Spiegel showed that, on the average, the onset of fatal symptoms was on the eleventh day with the limits at one to twenty-seven days. The average for the cases of Miller and Rogers[36] was also the eleventh day. Sixteen out of the nineteen embolic cases of Zurhelle occurred in the first and second weeks. In de Quervain's large statistics, there were two peaks in the incidence of embolism based on the interval between operation and death. The first peak occurred during the first week and the second in the middle of the second week. In Henderson's 267 verified cases, the average interval between operation and death was fourteen days. Seventy-five per cent. of Hampton and Wharton's cases occurred during the second and third weeks.

It is to be remembered that operation may loosen a pre-formed thrombus so that embolism, generally of the septic type, may occur at the time of the operative procedure. Abbott[69] stressed this danger when he stated that too little attention has been given to the thrombus which may be disturbed or infected by operations or may materially affect the prognosis of the operation. Cutler and Hunt[70] believe that small emboli can actually take place during

operation, as they have known patients to come out of the anæsthetic with chest pains, demonstrable friction rub and spitting up of blood-tinged sputum. They also believe that in the majority of cases, the various types of post-operative pulmonary complication are due to embolism from the operative field and not due to aspiration of infected material through the respiratory passages. Holman[71] found it impossible to produce a pulmonary abscess by the introduction of septic material into the bronchus. He concluded that post-operative pulmonary suppuration probably has its origin more often in the setting free of a septic embolus into the blood-stream by operative measures.

Death may be instantaneous after embolism or may not occur until sometime later. The duration of symptoms from onset of pulmonary symptoms to death varied from sudden to ten days, except in one patient who lingered on until the twenty-sixth day when the third shower of emboli carried him off. Twenty per cent. of the cases died suddenly. Sixty per cent. died between the first and third days.

Post-operative blood changes.—Allen[72] found in twelve patients a sharp increase of fibrinogen, a sharp prolongation of prothrombin time, and leucocytosis. The blood calcium showed only slight variations with a tendency toward a decrease. The number of platelets, the cholesterol, the bleeding time, and the coagulation time did not show definite changes. On the other hand, Andrews and Reuterskiold[73] after intensive post-operative blood-chemical studies for twenty-four and thirty-six hours after operation found no significant changes in the leucocytes, blood-pressure, temperature, pulse, blood-sugar, water content of the blood, chlorides or carbon dioxide. In every case, there was an enormous rise in the calcium in the blood, accompanied in all severe cases by an equally large fall in the potassium so that the potassium-calcium ratio often fell to below one. It is still unsettled as to how these blood changes may affect the incidence of post-operative thrombosis and embolism. Welch[74] could see no definite and constant relation between the amount of fibrin obtainable from the blood or the rapidity of its coagulation in the test tube and the occurrence of thrombus in human beings.

Influence of diet.—It has been suggested by Mills[75] and by Mills and Necheles[76] that the protein in the diet of post-operative patients may be an important etiological factor in the production of thrombosis, as the tendency to thrombosis appears during convalescence, usually shortly after the patient has begun to partake of a full diet. They found experimentally an increased coagulability of the blood following protein intake and lack of such effects with carbohydrate or fat. They observed that this marked shortening of blood-clotting time was intimately associated in time and degree with the specific dynamic action of protein. From the monographic work of Benedict and Carpenter,[77] it is well known that the specific dynamic action is greatest and more prolonged after protein ingestion than after carbohydrate or fat ingestion. It is interesting to note that this specific dynamic action effect with its increased cellular activity is manifested also by a more rapid healing of

wounds in dogs (Clark[78]), and in white rats (Harvey and Howes[79]) than with other foodstuffs; by a more rapid rate of blood-regeneration in dogs (Hooper and Whipple[80]); and by marked hypertrophy of the kidneys (Osborne and associates,[81] Jackson and Riggs,[82] and Smith and Moise[83]). By animal experimentation, Bancroft, Kugelmass and Stanley-Brown[84] found that the tendency to bleed or clot are definitely influenced by diet, and that lipins and globulins are the source of the blood-clotting substances, initially arising from the daily dietary.

Of the twenty-five post-operative cases in the series here reported, fourteen were on a general diet at the time of onset of symptoms, eight on a soft diet, and three on liquids. All those on soft diets and liquids were given plenty of custards, ice cream, malted or plain milk, egg nog, beef or clam broth, and orange albumin—all rich in proteins or protein derivatives. The protein in the diet may have some etiologic relationship to post-operative thrombosis and embolism, but cannot be considered as a dominant factor since all post-operative patients in this hospital are given relatively the same type of diet and the incidence of thrombosis in these patients is very low.

Influence of obesity.—Obesity is generally believed to predispose to thrombotic and embolic phenomena. Snell[85] found that of the 156 post-operative deaths in obese persons, forty or 25.6 per cent. were due to fatal embolism and that pulmonary embolism was about three times as frequent a cause of death in the obese group as in the control group. However, the average age of his obese group was fifty-five, so that the age factor strongly comes into consideration. In the cases here reported, seven of the twenty-five post-operative embolic deaths were in definitely obese patients; but all seven cases were above fifty years old, ranging from fifty-three to seventy. Extensive, well-controlled statistics will be necessary before any definite etiologic importance can be attached to obesity.

Influence of exertion in setting free a thrombus.—One often observes the dramatic suddenness with which embolism may make itself evident, especially about the time when the patient is allowed out of bed. It is to be observed that in our series, 64 per cent. of the cases occurred in the first and second weeks post-operatively: in other words, these patients have been sitting up in bed, in a wheel-chair, or up and about the wards. Fatal embolism can occur during sleep. One patient who had spent a comfortable evening and morning, suddenly woke up from an afternoon nap with extreme dyspnœa, rapidly became unconscious, and died ten minutes later. Another patient awoke with a scream, called for air, became cold and clammy, and expired. Farr and Spiegel profess skepticism as to any essential significance of straining at stool, sitting up in bed, and other types of exertion in precipitating embolism, because they found in several instances the obvious age of the pulmonary embolus precluded such interpretation.

Infarction.—We know that not all cases of pulmonary embolism are associated with infarction. Welch, in 1899, stated that hæmorrhagic infarction occurs during broken compensation of cardiac disease, weakness of the right

heart, fatty degeneration of the heart, general feebleness of the circulation, pulmonary emphysema and infective diseases. Welch and Mall[86, 87] showed experimentally that given the proper degree of stagnation, the greater the capillary blood-pressure, the more rapid is the infarction and the greater is its intensity. In their extensive studies on infarction, Karsner and Ash[88] experimentally found that hæmorrhagic infarction takes place only by slowing the circulation considerably as by the ligation of the pulmonary vein or by compressing the lungs by artificial effusion and that the greater the degree of stasis, the sooner is the true infarction likely to appear. In the Albany series, there were twelve cases of infarction among the twenty-five post-operative cases of pulmonary embolism. In nine of these twelve patients, the heart weight varied from 330 to 500 grams. In one patient, where the heart weighed 285 grams, there was quite a marked passive congestion of the lungs and abdominal viscera. In another with a heart weight of 230 grams, there were extreme congestion and œdema of the lungs. Three showed cardiosclerosis, two chronic vegetative endocarditis of the aortic valves, five chronic passive congestion of the viscera, three œdema of the lungs, three acute bronchopneumonia, one chronic fibroid phthisis, and one septicæmia. Of the remaining thirteen cases of post-operative embolism without infarction, the heart weight varied from 270 to 470 grams, of which three showed focal toxic myocarditis and two acute bronchopneumonia but no evidence of marked pulmonary circulatory stasis. Apropos of pneumonias, Hedinger and Christ[66] strongly stress the point that one should consider the possibility of a hæmorrhagic infarction being the underlying factor in pneumonias of the aged.

Symptomatology of embolism.—Death from pulmonary embolism may be of dramatic suddenness. A patient sitting quietly in a wheel-chair or pleasantly conversing with other patients or relatives may suddenly fall over dead without warning. Others may show a sudden onset of extreme dyspnœa, rapidly become unconscious and expire a few minutes after the onset of symptoms. Still others may show a stormy but extremely rapid course—sudden screaming, calling loudly for air, becoming cold and clammy, and expiring immediately thereafter. In these cases of sudden death, necropsy showed complete occlusion of the main pulmonary artery or of both right and left branches. Fig. 3 illustrates very well the completely occluding type of large embolus, extending from the right ventricle into the main pulmonary artery.

Where death was delayed from several hours to ten days, the symptomatology varied from the more quiet to the violent type. In the former, the patient was somewhat restless and sleepless, followed by periods of listlessness and weakness; was very pale and perspired freely. The skin was cold and clammy. There might be vomiting of heavy thick fluid with or without nausea and passing of flatus, or expectoration of blood-tinged sputum. Towards the end, the breathing became labored. Often there was cyanosis. The pulse could not be felt and finally the breathing stopped. In

the violent type, the patients were very restless and delirious, talking very irrationally. Sometimes they became violent—screaming loudly, sitting up in bed and fighting madly. After several such attacks of violence at short intervals, they were exhausted and quiet except for twitching of the limbs. There was profuse perspiration. The eyes took on a glassy appearance. The

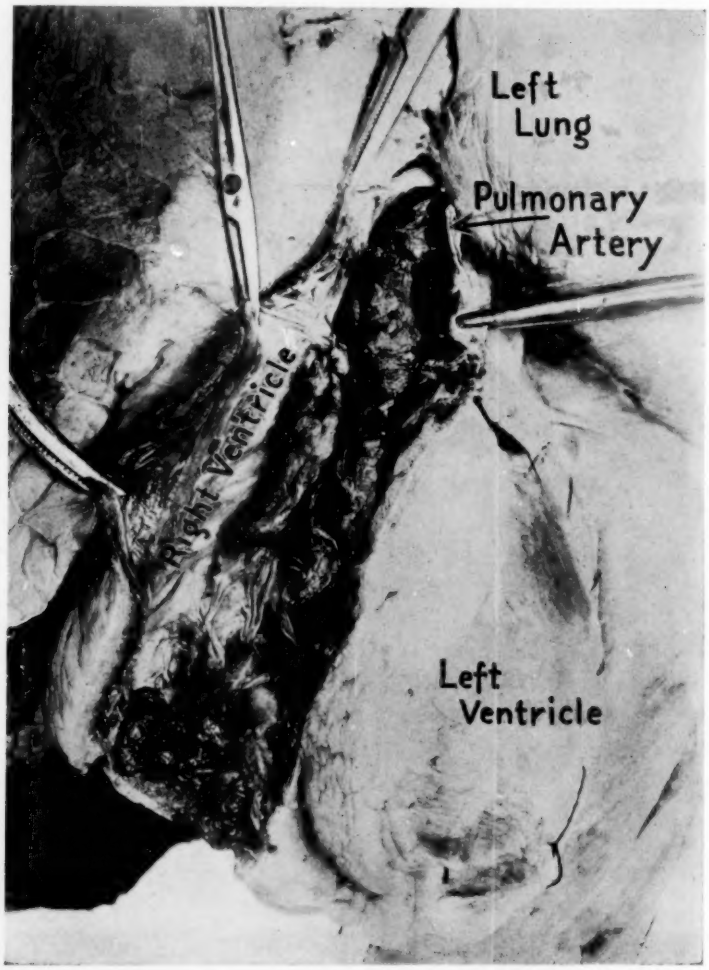

FIG. 3.—Photograph of heart with the right ventricle opened to show an embolus extending through the pulmonary valve into the pulmonary artery. Note that the lower end of the embolus is turned on itself, thus completely occluding the pulmonary orifice. The bend in the embolus has been partially broken, due to post-mortem handling.

pulse was weak and thready, and finally could not be felt. There might be a period of unconsciousness and Cheyne-Stokes breathing preceding exitus.

The symptoms in order of their frequency were as follows: Shortness or difficulty of breathing, restlessness, cyanosis, profuse perspiration, vomiting, pains in the chest, coughing with or without blood-tinged expectoration, delirium. The pains in the chest were found in those cases where necropsy

showed infarction with or without an associated bronchopneumonia. Seven of the twelve cases of infarction did not have chest pains. Here the infarctions were smaller or more centrally located so that the pleura was not implicated. Miller[89] in 1902, called attention to the significance of this post-operative pleurisy in its relation to pulmonary embolism. In one case in the Albany group where a marsupialization was done for an enormous pancreatic cyst and where the patient complained of severe chest pains, necropsy showed no pulmonary or pleural lesions but widespread fat necrosis in the abdominal cavity. Where cough was present, the lungs showed purulent bronchitis, broncho-pneumonia or diffuse pneumonitis.

The clinical history may indicate that the patient is having showers of emboli. The intervals between these showers varied from a few minutes to many days in the cases here reported. One patient had spent the last four days comfortably in a wheel-chair. One evening he suddenly became white and speechless, and perspired freely. His face was corpse-like with bulging eyes and moderately dilated pupils. He answered questions intelligently. Pulse was irregular and the heart sounds could not be heard. Face was cyanotic. There was air hunger. Wheezing and râles were heard throughout both lungs. He complained of pain over the precordium. After five minutes, the pulse became more regular and the patient began to feel more comfortable. Ten minutes later, he had another attack and died almost immediately. Necropsy showed the right pulmonary artery completely plugged. In these cases, multiple emboli both recent and old may be found scattered throughout the lungs or only one large embolus. In the latter case, the sequence of events suggests that by the force of the pulmonary stream, the different coils of the large but only partially occluding embolus are either pushed into the remaining aperture or the embolus might grow larger by secondary thrombosis.

EMBOLISM IN TRAUMATIC CASES

There were only three cases in which embolism occurred in traumatic cases.

CASE I.—H. F., male, seventy-one years old, obese, was struck and knocked down by an automobile. There were numerous contusions and abrasions but no fractures and no cerebral injuries. After a week in bed, he was allowed up in a wheel-chair. On the eleventh day after admission, while talking and joking with other patients, he suddenly collapsed and died in a few minutes. Necropsy showed extensive hæmorrhage into the retroperitoneal tissues, cardiosclerosis with hypertrophy (heart weighed 510 grams), embolism of the main pulmonary artery, and thrombosis of varicose veins of right leg, right femoral and right iliac veins. Other findings were acute gastritis, cholelithiasis, and focal necroses of the liver.

CASE II.—D. C., male, seventeen years old, accidentally shot himself while hunting, the bullet grazing his left leg and entering his abdomen. .Operation consisted in brief of repair of gunshot perforations of the stomach and suture of laceration of the liver. He improved satisfactorily for about a week and then rapidly began to lose ground. Empyæma of the left chest developed for which a thoracotomy was done. For two months, there was a profuse drainage from this chest. Eighteen days before death, gastric contents were detected in the empyæma discharge. He became very weak,

delirious, had much respiratory difficulty and died ninety-seven days after admission. Necropsy showed thrombosed vessels leading to a hæmorrhagic infarction of the right upper lobe of the lung, and widespread suppurative process of the left pleural cavity, left lobe of the liver, stomach, kidneys, and heart. There was in addition a localized purulent peritonitis.

CASE III.—A. C., male, sixty-three years old. While hurrying, he tripped over a wire, and struck his chin with considerable force as he fell. He was able to get up and walk to his office but soon developed severe generalized pain in his head, and became irrational and amnesic. Next day, he was rational but developed diplopia. Four days later, he became semicomatose. Fundus examination of the eye showed no evidence of intracranial pressure. Nine days after admission, he developed cyanosis and irregular respirations before death. Necropsy revealed fractured skull with subdural hæmorrhages, pulmonary embolism with infarction of left upper lobe, left pulmonary tuberculosis, right bronchopneumonia, obliterative endarteritis of the coronary vessels, thrombosis of coronary artery with infarction, and acute glomerular nephritis.

It may be that Cases I and III should be classified with medical embolism and Case II with post-operative embolism, but trauma may have been the contributing factor which aggravated the thrombosis or precipitated the embolism. It is to be noted that death occurred nine, eleven, and ninety-seven days after the accidents. Embolism in strictly traumatic cases was found to occur more commonly late by Strauss[90] and McCartney[91] after the usual time of occurrence of post-operative embolism. Most of McCartney's cases had fractures, only one of which was compound in type. Where the source of embolus was found, the thrombus was located at or near the site of injury. It is to be remembered that fat embolism is another very important complication of fracture that one must differentiate from embolism following thrombosis. At the Albany Hospital, there have been a number of cases of fat embolism following fractures, two of which were studied in great detail by Elting and Martin.[92] Moreover, fat embolism may be associated with embolism due to thrombosis. In one of the post-operative cases of embolism in the Albany series where the patient who was extremely obese had performed on her a ventral herniotomy with lipectomy, fat embolism was found associated with thrombosis and embolism as shown by the presence of fat globules in the meshes of the embolus after Scharlach R stain. For details on the diagnosis and pathology of fat embolism, the reader is referred to the excellent articles of Warthin,[93] and Elting and Martin.

MEDICAL EMBOLISM

The wide distribution of cases of medical embolism is shown in Table VI. There were twenty-one men to fifteen women, or 1.4 : 1. Seventy-eight per cent. of the cases occurred between the ages of forty-one and seventy. There was only one child in this group—a boy of eight who had a hæmolytic staphylococcus septicæmia with multiple septic infarctions of the lungs and septic lesions of the heart, brain and leptomeninges, caused by bacterial emboli. The primary foci in this case were the sphenoidal and ethmoidal cells. Medical embolism in children appears to be quite uncommon. Smellie[94] believed his case of pulmonary embolism with infarction of the right lower lobe in a

TABLE VI

Necropsied Cases of Medical Pulmonary Embolism

No.	Sex	Age	Diagnosis	Weight of heart—gms.	Source of embolus	Pulmonary infarction—lobes
1	M	41	Cardiac hypertrophy with dilatation (decompensation)	...	Undetermined	Right lower
2	F	42	Pyæmia, empyæma, peritonitis	300	Pelvic veins	Multiple, septic
3	M	68	Nephrosclerosis with uræmia	750	Undetermined	Right and left lower
4	M	64	Chronic vegetative endocarditis of tricuspid, mitral and aortic valves	360	Tricuspid valves	Left lower Right upper, septic
5	F	56	Paralysis agitans, bronchopneumonia	240	Undetermined	Multiple
6	M	51	Chronic endocarditis of tricuspid and mitral valves, mural thrombus in left ventricle	740	Prostatic plexus	Right lower and middle Left lower
7	F	42	Influenza, subacute meningo-encephalitis	...	Undetermined	Multiple septic
8	M	41	Hodgkin's disease	280	Undetermined	Right lower and middle Left lower
9	M	31	Subacute and chronic vegetative endocarditis of tricuspid, mitral and aortic valves	700	Tricuspid valves	Multiple septic
10	F	23	Streptococcic sore throat followed by bronchopneumonia and empyæma	205	Right iliac vein and inferior vena cava	Septic, right and left lower
11	F	48	Streptococcus hæmolyticus septicæmia	415	Veins of broad ligament, left ovarian vein	Multiple septic
12	M	57	Bronchopneumonia, pleurisy with effusion	385	Undetermined	Left lower
13	F	22	Subacute and chronic vegetative endocarditis of mitral valve, streptococcus hæmolyticus septicæmia	400	Undetermined	Septic, right lower
14	F	59	Primary carcinoma of left ovary with widespread metastasis	250	Undetermined (tumor thrombus)	None
15	F	78	Aortic aneurism, mural thrombi in right ventricle	470	Right ventricle	Right lower and left upper
16	M	Old	Parkinson's disease	...	Undetermined	Yes
17	F	68	Bilateral ovarian carcinoma with extensive peritoneal implantation, mural thrombus in right ventricle	...	Right ventricle	None

TABLE VI—(*Continued*)

No.	Sex	Age	Diagnosis	Weight of heart—gms.	Source of embolus	Pulmonary infarction—lobes
18	M	8	Chronic secondary anæmia, hæmolytic staphylococcus aureus septicæmia	110	Undetermined	Multiple septic
19	M	66	Multiple abscesses of both kidneys, aneurism of right internal iliac artery	310	Undetermined	Right lower
20	M	51	Acute on chronic glomerular nephritis	430	Renal veins	None
21	M	64	Acute intracapillary glomerular nephritis	390	Undetermined	Multiple
22	M	56	Cardiac infarction left ventricle, mural thrombi in right auricle	610	Right auricle	Left lower
23	M	55	Spongioblastoma multiforme	460	Undetermined	Right and left lower
24	M	65	Cardiac infarction left ventricle, mural thrombi in right auricle	670	Right auricle	Multiple in left lower
25	M	55	Tabes dorsalis	500	Vesical veins	Right and left lower
26	M	53	Pneumococcus endocarditis of tricuspid valve with Type I septicæmia	330	Tricuspid valves	Multiple septic
27	F	34	Rheumatic pancarditis with severe decompensation, acute bronchopneumonia	...	Left ovarian veins	Multiple, old and recent
28	F	67	Coronary thrombosis with apical infarction	640	Undetermined	Multiple
29	F	37	Chronic rheumatic pancarditis with decompensation, subacute pericarditis, acute intracapillary glomerulonephritis	540	Right auricle	Right and left lower
30	F	65	Cardiac infarction	410	Undetermined	None
31	M	44	Syphilis (gummas of liver, *etc.*)	615	Undetermined	None
32	M	68	Encephalomalacia, acute bronchopneumonia	540	Undetermined	Left lower
33	F	47	Toxic adenoma of thyroid, cardiac infarction	315	Undetermined	Left lower
34	M	59	Cardiac infarction	470	Undetermined	Right upper and lower Left lower
35	F	48	Marked thrombophlebitis of varicose veins of both lower extremities	330	Left iliac vein	None
36	M	65	Cardiac hypertrophy and dilatation (decompensation)	770	Undetermined	Left lower

boy of nine years was due to the streptococcus rheumaticus, causing early endocarditis of the right auricle. One and thirty-five hundredths per cent. of Gruber's and 1.83 per cent. of Rupp's embolic cases occurred below ten years of age. Only four of the Albany series were definitely obese, but these individuals were forty-eight to sixty-seven years old. Age appears to be an important factor in medical as well as post-operative embolism.

It is interesting to note that fifteen (42 per cent.) of the thirty-six medical cases were cardiac patients—six with cardiac infarction, four with cardiac decompensation, five with vegetative endocarditis, and two with rheumatic pancarditis. Except for three, the heart weights varied from 400 to 770 grams.

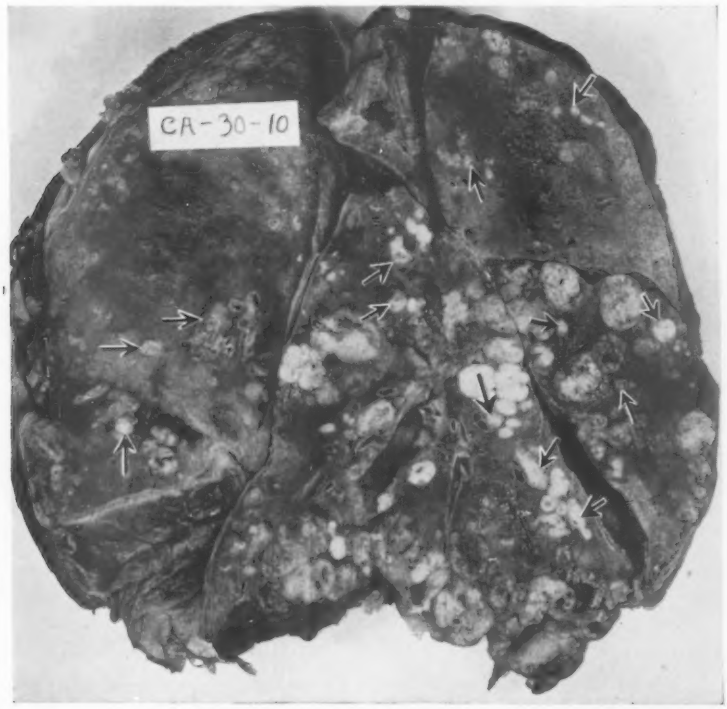

FIG. 4.—Photograph of lung in a very recent case of malignant endothelioma, primary in the gluteal region. Arrows point to some of the more noticeable smaller arteries plugged with tumor thrombi. To the extreme right of the photograph in the middle lobe, there is a small subpleural hæmorrhagic infarct.

Eighteen or 50 per cent. of the patients showed evidence of marked bacterial infection somewhere in the body.

Only six of these cases did not have infarction with the embolism. One of these was in a case of primary ovarian carcinoma with extensive peritoneal implantations and metastases. In the emboli in the pulmonary artery branches were groups of tumor cells. On the other hand, emboli may consist wholly of tumor cells. In a very recent case of malignant endothelioma primary in the gluteal region, there was a large, cauliflower-like, tumor thrombus growing beneath a leaflet of the tricuspid valve; in all lobes of the lungs, many of the

smaller branches of the pulmonary artery were occluded with tumor thrombi with only a small infarction resulting (Fig. 4). A large artery to the left lower lobe was plugged with friable tumor embolus. Warren[95] very recently observed a chondrosarcoma of the sacro-iliac synchondrosis with extension into the large veins of the pelvis and on up into the inferior vena cava. Nineteen days before death, a considerable portion of the tumor mass in the vena cava broke away and lodged in the left pulmonary artery where it continued to grow, producing a tree-like cast of the pulmonary blood-vessels and completely occluding the pulmonary artery in the process. Welch[67] stated that there have been instances of sudden death from blockage of the pulmonary artery by cancerous and sarcomatous emboli. Schmidt[96] reported several cases of gastric carcinoma where the smaller branches of the pulmonary artery were occluded with emboli of tumor cells resulting in hypertrophy of the right ventricle. Eschbach's[97] most unusual case of leiomyosarcoma arising beneath the endocardium just below the pulmonary valve produced an almost occluding tumor thrombus of the pulmonary artery, complete occlusion of the left pulmonary branch and incomplete occlusion of the right with a hæmorrhagic infarction of the right lower lobe of the lung and dry pleurisy. In Shennan's[98] case, a spindle-cell sarcoma of the mediastinum extended through the heart into the right auricle where the growth was so large as practically to occlude the tricuspid valve opening.

In the medical cases, infarction was very common after embolism. The lower lobes were involved in 64 per cent. of the cases with infarction—27 per cent. in the right lower and 37 per cent. in the left lower. Contrary to post-operative infarction, the medical infarction occurred more often in the left lower lobe of the lung. In 24 per cent., there were multiple infarctions, all lobes being involved. The upper and middle lobes were affected only five times but an infarction was found also in one of the lower lobes.

The symptomatology of embolism with or without infarction in medical cases was essentially not different from that seen in post-operative cases. From onset of embolic symptoms to death, the duration varied from sudden to twenty-seven days. About one-third of the patients died within the first three days, two of which were sudden deaths. One of these sudden deaths occurred during the quiet of a sleep from which the patient was awakened with extreme shortness of breath, and the second, during the exertion of being helped onto a bed-pan. Very frequently, it is extremely difficult to estimate the onset of embolism in medical cases. The severity of the symptoms of heart disease or of abdominal disease may mask the pulmonary picture, or the patient may be too ill to complain of added discomforts. On the other hand, the pulmonary symptomatology may clearly indicate that the patient is having a series of embolism at various intervals. Two cases in particular showed many showers of emboli when finally the patients died fifteen and seventeen weeks later. Showers are especially liable to occur in cases of vegetative endocarditis or mural thrombosis of the right side of the heart.

CONCLUSIONS

(1) Sixty-four verified cases of pulmonary embolism with or without infarction form the basis of this study. They consist of twenty-five post-operative, three post-traumatic, and thirty-six medical cases of embolism. This gives a necropsy incidence for embolism of 7.9 per cent., a mortality incidence of 2.1 per cent., and a morbidity incidence of 0.102 per cent.

(2) Sex has probably no etiologic significance. The age incidence of embolic cases suddenly increases from forty years onward. Our obese patients with embolism were all in this age liability group.

(3) Embolism occurred in 0.09 per cent. of the patients after general surgery and in 0.08 per cent. after gynæcological surgery. Sixty-four per cent. of the cases occurred in the first and second weeks post-operatively. The duration of symptoms from onset of pulmonary embolism to death varied from sudden to ten days, 80 per cent. dying by the third day. Sixty-eight per cent. of the operations were in the lower abdomen. On the other hand, in medical embolism, the duration of symptoms were longer (as long as twenty-seven days) due to the greater frequency of smaller emboli occurring sometimes in showers. Furthermore, patients with medical embolism were not so severely affected, since only about one-third of them died within the first three days.

(4) Forty-two per cent. of the post-operative emboli lodged in the lower lobes, more often on the right in the ratio of almost 2 : 1; 42 per cent. in the main pulmonary artery or in one or both of its two branches, the right being favored. Due to the frequency of the embolism being less massive in medical cases, the emboli were able to reach the smaller branches of the pulmonary artery; in 64 per cent. of the cases, the lower lobes were involved, but, contrary to post-operative infarction, medical infarction occurred more often in the left lower lobe than in the right lower.

(5) There was manifest infection in only 32 per cent. of the post-operative cases, and in 50 per cent. of the medical cases.

(6) Infarction after embolism is liable to occur when there is an added circulatory congestive disturbance. The heart showed a varied pathology of hypertrophy, cardiosclerosis, and endocarditis. The hearts in the medical cases were uniformly larger and 42 per cent. of them showed severe lesions of hypertrophy and dilatation (decompensation), coronary thrombosis with infarction, vegetative endocarditis, and pancarditis. Infarction occurred almost twice as frequently after medical embolism as after post-operative embolism.

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NERVE SUTURE AND MUSCLE REPAIR

PRIMARY SUTURE OF THE ULNAR NERVE AND SECONDARY RECONSTRUCTION OF THE EXTENSOR TENDONS OF THE FOREARM

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When badly lacerated wounds are treated in a well-equipped hospital immediately after a patient has been injured, frequently all the important anatomic structures can be repaired, and the patient need suffer no permanent disability. Upon admission to a hospital, an injured person, unless he is suffering from hæmorrhage or shock, can be quickly placed in a state of light anæsthesia, when the occasion requires it, for the treatment of his wound. The wound can be thoroughly cleansed of all extraneous particles, and the devitalized tissue carefully removed. An effort can be made to preserve all the tissues which have not been devitalized. Often structures which have been severed or lacerated can be repaired by suture, and the wound can be closed. Such wounds in the majority of cases heal by primary union. A wound should be drained when its surfaces cannot be approximated and when there is a likelihood of dead spaces forming that will allow the accumulation of blood or serum, which makes a fertile medium for the growth of the bacteria with which the wound was contaminated. If it is considered necessary to drain the wound, adequate drainage should be established to facilitate the rapid escape of blood and serum from the depths of the wound. Badly contaminated wounds are usually better left open and an antiseptic treatment carried out. When a primary closure of a wound is feasible, the wound should be thoroughly swabbed with an antiseptic solution. Whether the wound is closed by primary suture, secondary suture, or allowed to remain open, the patient should be given a prophylactic dose of tetanus and gas gangrene antitoxin.

The following case illustrates the results which may be obtained when the treatment of badly lacerated wounds is carried out under favorable conditions.

Case No. 1325.—V. E. M., aged fourteen years, was brought into Casualty Hospital, Washington, D. C., August 22, 1922, immediately following an injury which occurred on a farm about ten miles from the city. The injury resulted in multiple lacerations of both arms and both legs. The boy had been driving a double team of horses hitched to a mowing machine. As he stood on the machine and leaned forward to adjust part of the harness, the horses became frightened and ran away. The machine suddenly lurched forward, and the boy lost his balance, fell forward, and dropped on the ground in front of the mower. As the machine passed over him the blades made several deep lacerations in his arms and legs.

When the patient was admitted to the hospital it was found that the shock and the loss of blood were not great enough to contra-indicate the immediate treatment of his

wounds. A state of light anæsthesia was produced by nitrous oxide, oxygen, and ether, and the wounds were treated.

The lacerations which had produced the most serious damage were those in the right upper extremity. On the posterior aspect of the right arm about two inches above the olecranon there was a deep laceration which had divided all of the tissues of the arm down to the humerus. When this laceration was explored it was found that the triceps had been partially severed, the ulnar nerve divided, and a groove furrowed in the

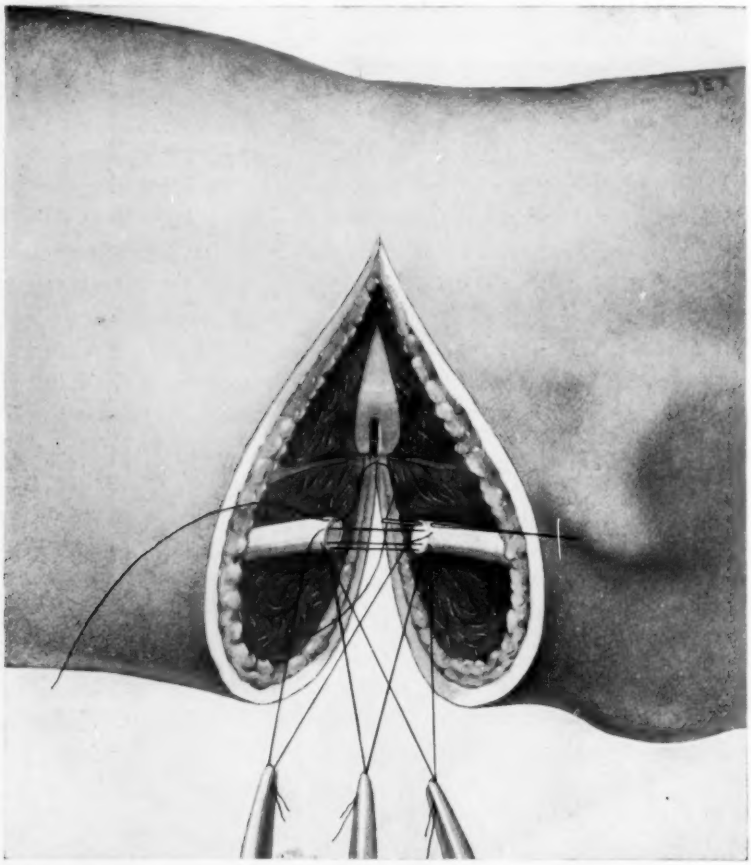

FIG. 1.—The end-to-end anastomosis of the ulnar nerve, showing the cut ends of the nerve and the method of placing the fine silk sutures in the sheath of the nerve. The laceration of the triceps and the groove that was cut in the posterior surface of the humerus are also shown.

humerus. This wound was bathed with ether, the devitalized tissues were excised, a 5 per cent. solution of picric acid was applied to the wound surfaces, and the tissues were approximated. The triceps was sutured by approximating the muscle fibres with plain catgut sutures, and the fascial sheath with interrupted chromic catgut sutures. The ends of the ulnar nerve had retracted. Both the distal and proximal ends were found. Each end was then cut squarely across, and these freshened ends were anastomosed with interrupted sutures of fine silk placed in the sheath of the nerve (Fig. 1). The skin was closed with silkworm sutures and silk. In the middle of the forearm there were two deeply lacerated incisions which had severed the superficial group of the extensor muscles in two places. These two wounds were washed with ether, and the devitalized skin, subcutaneous fat and muscle were excised. The débridement required the removal of the

superficial group of extensor muscles which lay loosely between the two lacerations. Picric acid in 5 per cent. solution was applied to the wound surfaces, and the wound was closed without drainage. Because of the number of lacerations and the time it required to treat them and to anastomose the ulnar nerve, it was thought not advisable to attempt a primary reconstruction of the extensor muscles of the forearm. The reconstruction of these muscles was therefore deferred. To immobilize the arm following the anastomosis of the ulnar nerve and the suturing of the triceps muscle, the arm, the forearm, and the

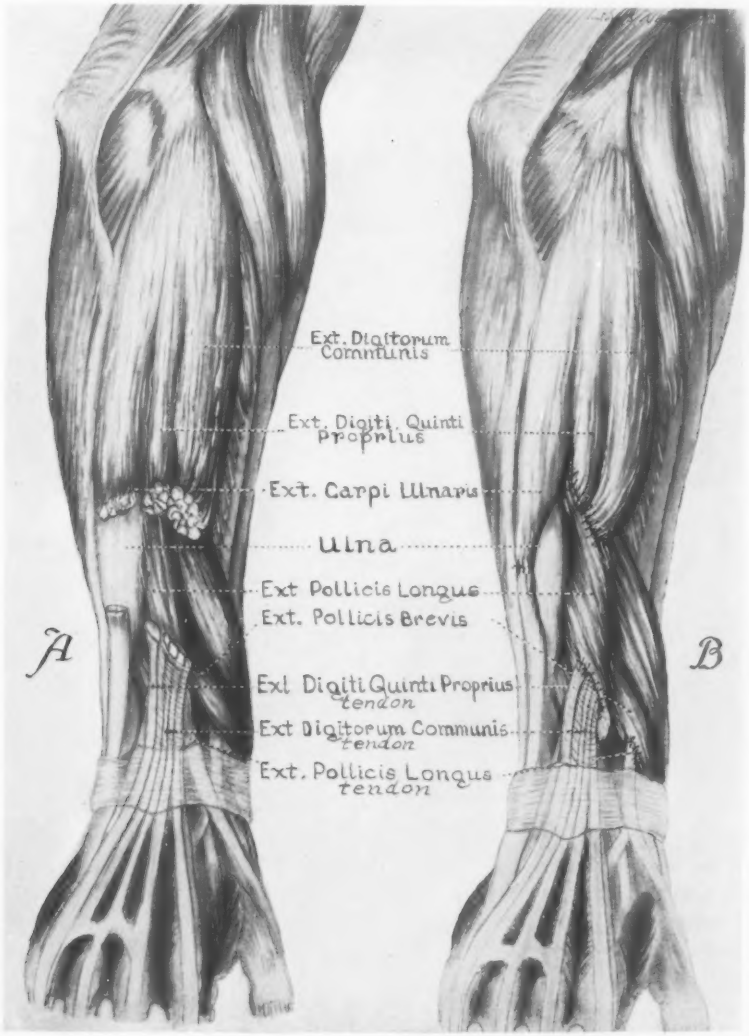

FIG. 2.—*A*—Showing the defect in the superficial extensor muscles resulting from the excision of devitalized muscle caused by a contused laceration. *B*—Showing the method employed to reconstruct the extensor muscles of the forearm.

hand were placed in a plaster cast. The wounds of the left forearm and the right and left thighs were bathed with ether, the devitalized tissues were removed, and the wounds were swabbed with 5 per cent. picric-acid solution and closed without drainage. A prophylactic injection of tetanus antitoxin was administered. All of the wounds healed by primary union. The patient was dismissed from the hospital September 1, 1922, and

he was instructed to return in about one month for a reconstruction of the tendons of the right forearm.

The patient was re-admitted to the hospital October 4, 1922, The following morning a reconstructive operation was performed. Through a long incision on the dorsal aspect of the right forearm the retracted ends of the divided extensor digitorum communis, extensor digiti quinti proprius, and extensor carpi ulnaris were found to be firmly held in scar tissue. The ends were dissected free from the scar tissue, and the reconstructive operation was planned. The proximal ends of the extensor digitorum communis and the extensor digiti quinti proprius were fastened into the belly of the extensor pollicis longus. The tendon of the extensor pollicis longus was divided about one inch above the wrist, and into it were sutured the tendons of the extensor digitorum communis and extensor digiti quinti proprius. The distal end of the extensor pollicis longus was sutured into the side of the extensor indicis proprius. The ends of the extensor carpi ulnaris were approximated by lengthening the tendon of the distal segment and suturing it to the end of the proximal segment (Fig. 2). Chromic catgut

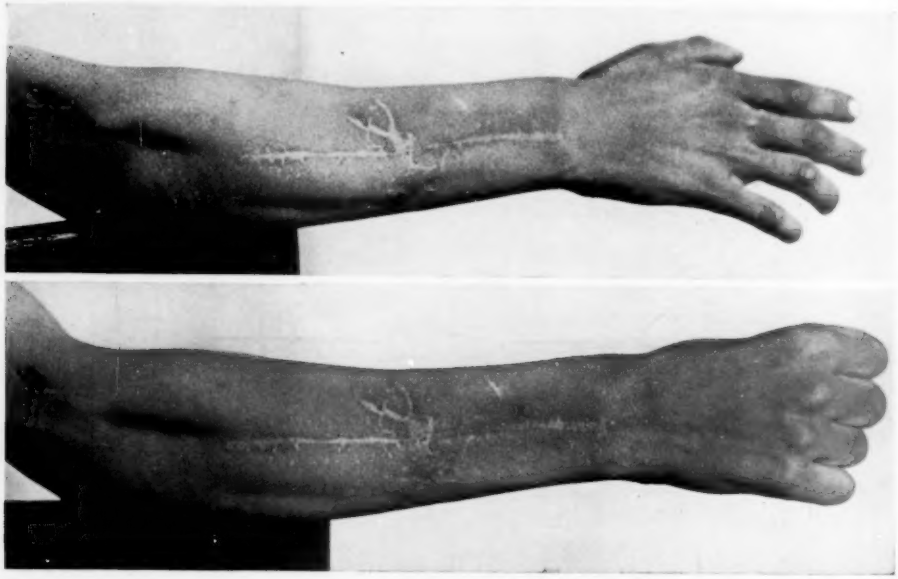

FIG. 3.—Photographs of the patient's right forearm and hand, showing the degree to which the patient can extend and flex the fingers.

sutures were used in the tendons. The wound was closed without drainage, and a splint was applied to the forearm. The wound healed by primary union.

The patient was kept under close observation so that I could note the return of the function of the ulnar nerve and of the extensor muscles of the right forearm. About one month after the reconstructive operation on the extensor muscles the use of the splint was wholly discontinued, and the patient was allowed to begin active movements. Both active and passive movements were gradually increased, and the function of the forearm and hand returned rapidly. Complete sensation came back slowly. Six months after the end-to-end anastomosis of the ulnar nerve, the patient had good sensation to cotton-wool and pin pricks over the ulnar side of the hand, the outer half of the middle finger, and the fourth and fifth fingers. Shortly after the return of sensation the patient started to play ball and continued to play it all spring and summer. A year and a half after his injury he played an entire season on a high-school baseball team. After leaving school he learned carpentry and has since worked at this trade. On April 13, 1931, I examined the patient and found that with the exception of a loss of sensation in the tip

of the little finger and an inability to completely extend the fourth and fifth fingers, the function of his right hand is entirely normal (Fig. 3). He now has a useful and almost completely normal hand.

DISCUSSION.—*Suture of the Ulnar Nerve.*—A favorable prognosis for the return of motor and sensory function following the suture of peripheral nerves depends largely upon whether the anastomosis is made soon after the injury. Most of the large series of cases of nerve suture which have been published comprise cases treated during the War and include cases of both primary and secondary suture or of secondary suture only. On the basis of several series of secondary suture, Thorburn[1] estimated that good results followed secondary suture in "somewhere between one-third and two-thirds of all cases." His personal experience was that in a secondary suture "a perfect neurologic recovery is rarely, if ever, obtained." Tinel,[2] in 1917, expressed a more optimistic view. "At the present time," he said, "basing our opinion on a very large number of observations made since the beginning of the War, we are justified in affirming that the prognosis of peripheral-nerve lesions is, on the whole, favorable. Every peripheral nerve affected by traumatism tends to regenerate, provided the general condition of the patient enables him to contribute towards this restoration." Tinel[2] investigated the results following nerve suture or grafting in 108 cases, some of which were operated upon under favorable, others under unfavorable, conditions. He found that in 22 cases there was an almost complete return of function, in 72 cases there was more or less rapid progress being made towards a restoration of function, and in only 14 cases was there no sign of regeneration. "Early intervention," Tinel said, "does not appear to be an indispensable condition. . . . Nevertheless, there can be no doubt but that early sutures are followed by more rapid regeneration." Delagenière,[3] who had 245 cases of nerve suture under observation for two and a half years following anastomosis, reached a conclusion somewhat similar to Tinel's. "The earlier the nerve suture is performed," Delagenière stated, "the more rapid is the regeneration." Before the War he had made a number of nerve anastomoses immediately following injury, and in every case the anastomosis resulted in a functional cure.

When a nerve is sutured soon after its injury there is a minimum of resection necessary as the nerve has not become invaginated by progressive sclerosis; there is also a maximum of mobility and elasticity of the nerve, which facilitates a good approximation of the nerve ends. Immediate suture may save the patient months of treatment and delay in cure if the intervention is successful; if it is not successful, it has not endangered the ultimate result.

The prognosis for the ulnar nerve is less favorable than for other peripheral nerves. Thorburn,[1] when discussing the results obtained at the Grangethorpe Military Hospital, in Manchester, said: "The ulnar nerve is particularly disappointing and the recovery of voluntary power in the small intrinsic muscles of the hand was exceedingly poor." Platt and Bristow[4]

expressed a similar opinion. A good return of function following the immediate suture of the ulnar nerve has been reported in a few instances. Rawlence,[5] in 1920, reported a case of a soldier who was wounded in the elbow-joint by an exploding shell. Four hours later the wound was cleansed, the devitalized tissues were excised, and the ends of the ulnar nerve "were brought together by a holding suture." An interrupted elbow splint was applied. Five days later the entire surfaces were cleansed, and the elbow-joint was sutured. The ends of the ulnar nerve which had been brought together were found in apposition. The silk holding-suture was removed and a flap of muscle from the internal condyle was brought over the nerve anastomosis. The arm was put in a splint in extension. Fifteen days later passive movements were started. After two more weeks the movements of the intrinsic muscles of the hand were good, although still weak. Two cases in which excellent results followed the suture of the ulnar nerve were reported by Lacroix,[6] in 1923. A boy, ten years of age, cut his right wrist, completely severing the ulnar and median nerves and the palmaris longus, the flexor carpi radialis, and the flexor digitorum sublimis tendons. Half an hour later the severed ends of the ulnar and median nerves were sutured end-to-end through the nerve sheath with interrupted stitches of No. 00 chromicized catgut. The tendons were sutured with silk. A dorsal splint was applied. Three weeks later resistive exercises were begun. Five weeks after the anastomoses were made, the gripping power and sensation were normal. Equally good results were reported by the same author in the case of a girl, nine years of age, who had the ulnar and median nerves and the palmaris longus, the flexor carpi radialis, the flexor carpi ulnaris, and the flexor digitorum sublimis tendons of the right wrist accidentally severed. The author used the same method of suture as he did in the preceding case. He reported that three and a half months after the injury the hand and fingers were normal. Bunnell[7] recorded two cases of a return of the sensory function following a suture of the ulnar nerve made some months after the injury in which it was severed. In one case all the nerves and tendons in the front of the wrist were destroyed by an infection which developed after the wrist had been cut. At operation, five months after the injury, the ends of the ulnar nerve were found to be an inch apart, and the ends of the median nerve three inches apart. The nerve ends were anastomosed by direct union, and the tendons indirectly by means of grafts. A little less than a year later all the fingers were sensitive to pin pricks and cotton-wool. In the other case reported by Bunnell the ulnar nerve was severed at the wrist. Twenty-one months later the nerve ends were sutured. Three and a half months after the anastomosis sensation was normal in the hand and fingers.

Suture of the Extensor Tendons of the Forearm.—The primary suture of tendons is a difficult and arduous procedure. But, as Lemaitre[8] pointed out, it gives results directly proportional to the care taken in examining the injured tissues; in excising all such devitalized tissues as might serve as culture media; in extracting all minute foreign bodies and fragments of

bone; in excising sparingly the skin, connective tissue, fascia, and muscles which might form "chambers of attrition"; and in practising scrupulous hemostasis and asepsis.

Not every injured person can be subjected to the tedious procedure of a reconstructive operation and primary suture. Certain conditions may exist which will necessitate deferring the reconstructive measure or delaying the closure of the wound. The wound may be contaminated or infected, it may contain a zone of lymphœdema or of lymphangeitis, or gas gangrene may be present. It may be impossible to explore the entire wound or to extract all the minute extraneous fragments. Reconstructive measures may have to be deferred on account of the great number of wounds requiring treatment. If the patient is suffering from shock, hæmorrhage, fever, or alcoholism, primary suture may be inadvisable, depending upon the degree of the condition.

Good results following a secondary reconstruction of the extensor tendons of the arm and hand have been reported. Such a case was reported by Bradburn,[9] in 1922. The extensor digitorum communis and the extensor pollicis longus had been severed when the patient ran his hand through a window pane. The tendons were sutured by another surgeon. The patient developed an inability to extend his middle and ring fingers and the terminal phalanx of his thumb. When Bradburn operated three months after the injury he found that two and a half or three inches separated the proximal end of the extensor digitorum communis from the tendons of the ring and middle fingers. With a continuous suture of No. 9 silk thread, new tendons were made of four strands of the suture connecting the tendons of the middle finger, the ring finger, and the thumb to the severed end of the extensor digitorum communis. Each silk cable was surrounded by a tube of fascia lata which was sutured to the tendon ends. The hand became normal in function, the new extensor longus pollicis moving independently of the middle and ring-finger tendons. A case was reported by Merrill[10] in which a soldier who was wounded on the dorsal surface of the left forearm could not extend his fingers and thumb after the débridement, suppuration, and healing of his wound. At operation, six months after the wound had healed, the stumps of the extensor tendons were found to be irregularly severed. A reconstructive operation on the extensor tendons was then carried out. After a period in which the hand was kept in a splint and a second period in which it was given exercise, the patient could extend his fingers and thumb normally and could move his index finger independently of the other three. In a case reported by Bunnell,[11] the extensor tendons of the index and middle fingers and the extensor carpi radialis brevis had been severed. Infection developed, resulting in scar tissue which bound all the dorsal tendons. Two months after the injury these tendons were dissected from the enveloping scar tissue. The severed ends of the extensor carpi radialis brevis were sutured directly. The divided ends of the extensor tendons of the index and long fingers were anastomosed indirectly by means of grafts taken from the

palmaris longus. Four months later there was a considerably improved but not wholly normal function of the sutured tendons. Bunnell[11] also reconstructed an extensor pollicis longus tendon three months after it had been accidentally severed. He used a graft of two and a half inches taken from the palmaris longus to bridge a gap between the divided ends of the tendon. Three months after the reconstructive operation the patient had an almost normal use of his thumb. Kanavel[12] mentions a case in which a destroyed portion of an extensor pollicis longus was replaced by strands of silk covered with a transplant of fat. Normal function of the thumb was restored.

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ANTIVIRUS TREATMENT OF MALIGNANT ŒDEMA INFECTIONS

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Infections of the human body caused by the bacillus of malignant œdema are so seldom met with these days that it seems advisable to report the following case, the more so because of the methods used in treating it and the results obtained.

The clostridium of malignant œdema, also called bacillus of malignant œdema—Koch or Vibrion Septique—Pasteur, is a large rod-shaped organism with rounded ends. Young bacilli retain the Gram stain—older ones do not. They are motile and possess many flagella. Endospores are formed, mostly centrally situated, sometimes at the end. The bacillus does not produce any proteolytic enzymes. It decomposes starch and carbohydrates, forming butyric acid, carbon dioxide, hydrogen sulphide and methane. The organisms are found frequently in the soil, hay dust and in the intestinal tract of the herbivora.

Simple inoculation of an abraded surface will not, as a rule, produce infection in either animal or man, because the presence of oxygen is detrimental to its growth, but when the bacillus is introduced into a wound and oxygen excluded, infection occurs.

The bacilli in the subcutaneous tissues produce marked œdema, with the formation of gases, which have an obnoxious odor. The muscles are rapidly destroyed by the organisms, but the tendons, nerves, cartilage and osseous structures do not seem to be affected. In fatal cases the organisms get into the blood-stream and attack the viscera.

Report of Case.—Leonard M., aged twelve years, white, fell from a tree, a distance of thirty-two feet, September 13, 1930, at 7:30 A.M. About an hour later he was seen by Doctor Merten who had him taken to the Milwaukee Hospital. When admitted the boy appeared to be in great pain, the face was pale, temperature, 99.2° Fahrenheit; pulse, 80; respiration, 20. The radius of the right arm was protruding two and one-half inches. Fifteen hundred units of antitetanic serum were given. At 9:30 A.M. the boy was placed on the operating table, put under ether anæsthesia, the wound was enlarged and it was found that there was also a fracture of the ulna, one-half inch from the joint, and that the radius had separated from its epiphysis. Fragments of dried leaves and grass were cleaned out of the wound. The wound was then washed out three times with ether, the bones put in apposition, a gutta-percha drain inserted, and the wound was closed. A splint was applied and the boy was sent back to bed. On the next day the temperature rose to 103°F. and the pulse to 100, the temperature declining to 102°F. by four o'clock in the afternoon. On the third day the arm was swollen up to the axilla, and an offensive odor was noticed. However, no crepitus could be elicited. Smears and cultures were taken from the wound by Doctor Oesterlin, who reported

that the smears showed the presence of a large bacillus which did not take the Gram stain.

The wound was then laid wide open and tubes were inserted for the continuous irrigation with Dakin's solution. The temperature dropped to 99.5°F., but the pulse remained around 100. No change was noted in the swelling of the arm.

On the eighth post-operative day, the report of a definite infection with the bacillus of malignant œdema was received from the laboratory. Because there was no vaccine or serum against bacillus of malignant œdema available, patient was given 10 cubic centimetres of polyvalent perfringens serum; this was followed by a chill and a rise in temperature; the next morning the temperature had dropped to 99.6°F. That day patient was given another 10 cubic centimetres of perfringens serum; no chill followed but the temperature rose to 101.2°F., but on the following morning dropped to 98.6°F.; the pulse, however, remained around 110.

The swelling of the arm not having receded much, the next day the boy was given

FIG. 1.

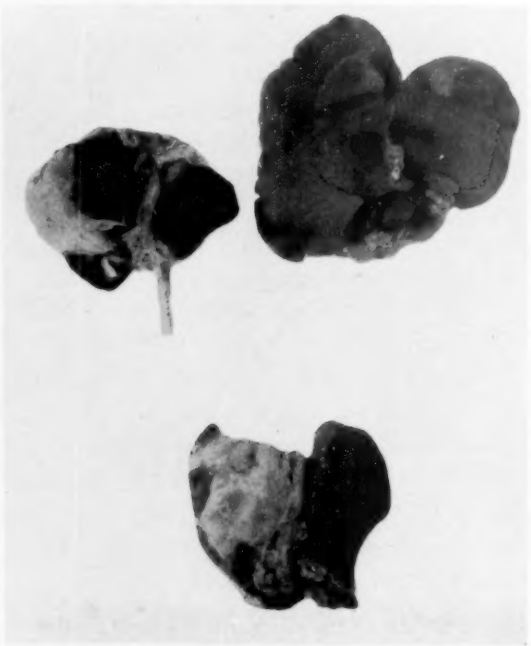

FIG. 2.

FIG. 1.—Lungs and liver of guinea-pig immunized by intrapleural injections of malignant œdema antivirus. Pleura smooth and shiny; positively no exudate. Liver shows no pathologic lesions.
FIG. 2.—Shows lungs of two control animals. They are covered by thick masses of fibrino-purulent exudate. Liver shows intense fatty degeneration.

20 cubic centimetres of perfringens serum. No chill followed, the temperature rose to 102°F., but by the following morning had dropped to 100°F., where it remained for about five days.

The swelling of the arm had receded somewhat and crepitation could now be elicited. We were unable to determine the presence of air bubbles by means of X-ray examination.

The temperature reached normal on the fifteenth post-operative day, and after that it fluctuated between 98.6°F. and 100°F., with the pulse between 100 and 120. Cultures taken at different times still showed the presence of the bacillus of malignant œdema. Four weeks after the injury, the arm was still swollen, and gas bubbles were still coming out of the wound.

On the twenty-eighth post-operative day, the Dakin's solution was discontinued,

and an antivirus (prepared by Doctor Oesterlin), was used as a dressing. The preparation of this antivirus will be described later. Within two hours after the antivirus had been applied to the wound, the temperature rose to 101.4°F. followed by a drop to normal within forty-eight hours. For the first ten days the wound was dressed every twelve hours with the antivirus preparation. At the end of thirty-six hours after the application of the antivirus, the swelling of the arm rapidly receded and after the third day had disappeared entirely. The wound itself now began to look cleaner and the amount of gas emanating from the wound was lessened. On the eighth day after the beginning of the antivirus treatment, the wound showed definite signs of healing and no gas could be found. The temperature fluctuated between 98.6°F., until the thirty-seventh post-operative day, when it remained normal until the patient's discharge from the hospital October 31, 1930. While the anatomic result in this case is not all that could be desired, the boy has a functional hand, and not an artificial appliance. At no time during the stay in the hospital did cultures or smears reveal any other organisms but the bacillus of malignant œdema.

Experiences with Filtrates of Bacillus Œdematis Maligni.—In our case no satisfactory success with the polyvalent serum could be obtained, and therefore Doctor Oesterlin tried to prepare an antivirus and to determine what effect this treatment might have in animals.

Already, in 1924, Besredka incubated broth cultures of staphylococci and streptococci for eight days at 37°C., then filtered them through Chamberland candles. He obtained atoxic filtrates which he called "Antivirus." It contains products of metabolism of the bacteria which act upon tissues producing a local immunity of the receptive cells in such a way that the cells are not sensitive any more to the virus and therefore the tissues become resistant to further infection.

Besredka performed the immunization intracutaneously, either by injection or by application of wet dressings with antivirus. He found the antivirus strictly specific, that means a previous treatment with staphylococcus antivirus only protected against a staphylococcus infection and not against streptococcus infection and *vice versa.*

The antivirus cannot be compared with any immune bodies like antitoxins, agglutinins, amboceptors, *etc.*, because of its thermo resistance. It can withstand a temperature of 100°C. for ten minutes.

In 1929, Doctor Oesterlin had demonstrated the action of antivirus in the pleural cavity. He injected filtrates of eight-day-old pyocyaneus cultures into the pleural cavity of rabbits and guinea-pigs and could immunize them against fatal doses of bacillus pyocyaneus. If he injected another antivirus or plain broth instead of pyocyaneus antivirus, the animals died the same as the controls which had not been treated previously.

Obtaining a strain of malignant œdema from our patient, we tried first to protect white rats by intrapleural injections of specific antivirus against malignant œdema infection. The antivirus was prepared in the following way: We made a broth culture of bacillus of malignant œdema in a flask, overlaying it with liquid paraffin and left it in the incubator for eight days; then we filtered the culture through a Berkefeld filter. We heated the filtrate five minutes to 100°C. Using this filtrate immediately, we had the same

effect as when we planted the filtrate once more with bacillus of malignant œdema, incubated it for eight days more and filtrated it a second time; therefore we used mostly "first filtrates." The antivirus proved to be perfectly innocuous.

The following is one of the many experiments which demonstrate the effect of the antivirus. We injected 2 cubic centimetres of malignant œdema antivirus into two rats intrapleurally and repeated the injection after three days. At the same time we treated one rat in the same way, using, instead of antivirus, plain broth. Five days later these three animals, and two new ones as control animals, were given 1 cubic centimetre of anaërobic broth culture of malignant œdema intrapleurally. The animal treated with plain broth and the two untreated animals died within twenty-four hours. They showed a severe pleurisy, both pleuræ being covered by fibrinopurulent exudate and the pleural cavity filled with 3 cubic centimetres of serohæmorrhagic fluid; there was also a fibrinopurulent pericarditis present.

The two animals treated with antivirus continued to be in perfect health. One of these animals was killed at the end of eight days and the other one after two weeks. They showed positively no lesions. The pleura proved to be free and perfectly clear and shiny, no exudate in the pleural cavity. No pneumonia.

In another set of experiments we tried to find out whether the action of antivirus is a specific one or not. We injected malignant œdema antivirus intrapleurally into two animals, bacterium coli antivirus into the third animal. The injections were repeated after three days. Two untreated animals were taken as controls. Then we injected 1 cubic centimetre of anaërobic broth culture of malignant œdema into all five animals. The animal treated with bacterium coli antivirus died at the same time as the control animals—twenty-four hours after injection, while the animals treated with malignant œdema antivirus were perfectly well after injection. After one week one animal was killed, the other animal after two weeks. The autopsy did not reveal any pathologic lesions in the thoracic cavity.

The results were similar after intracutaneous injection of antivirus. We made experiments in guinea-pigs and white rats. We injected twice intracutaneously 5 cubic centimetres of malignant œdema antivirus. As control we again used animals injected with the same amount of coli antivirus and untreated animals. All these control animals died within twenty-four hours. They showed the typical picture of malignant œdema: the skin was discolored greenish-black, the abdominal wall very œdematous; the muscle was very brittle and peeled readily from the skin.

The immunized animals were, however, not without lesions.

The effect of the injection was not a diffuse phlegmon, but a circumscribed infiltration of variable size; later on an ulcer developed which healed in thirty to forty days. In one case the subcutaneous injection in the centre of the abdominal wall produced no lesions at the site of injection, but an œdematous phlegmon of the skin of the thorax developed. The animal recovered.

It seems paradoxical that we were not able to obtain any results in animals with wet dressings with antivirus regardless whether we applied them before or after injection. All the animals died at the same time as the control animals.

SUMMARY

(1) A case of malignant œdema infection has been reported.

(2) Most of these cases require amputation to save life.

(3) The antivirus was the factor in saving the hand of this patient.

(4) Experiments in guinea-pigs and white rats showed a specific action

of malignant œdema antivirus previous to infection with the corresponding germ:

(*a*) When we applied the antivirus *intrapleurally, no* lesions occurred in the previously treated animals, while all the control animals died as well as the animals previously treated with *another* antivirus.

(*b*) Subcutaneous prophylactic treatment protected the animals against death, while the control animals as well as animals treated with a different antivirus died between twenty-four hours. Infection, however, *took place* in form of localized infiltrations which exulcerated and healed in about a month.

(5) Wet dressings with filtrate were of no avail in animals.

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CLOSING THE BRONCHIAL STUMP IN PULMONARY SURGERY*

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Permanent closure of the bronchial stump in performing a lobectomy is one of the factors of greatest importance. Failure to obtain a permanent air-tight stump is by far the greatest single factor responsible for the high mortality of this operation. Experimental studies have been reported elsewhere[1] which adequately explain the reason for lack of healing of the bronchial stump in a high percentage of cases. The experiments reported at this time describe a method in its various stages for the permanent closure of large bronchi.

Experimental.—Dogs were used exclusively. A pre-bronchoscopic dose of morphine gram 0.015 and atropine gram 0.0004 per kilogram of body weight was given about one and one-half to two hours before bronchoscopy. Five, 15, 35, 50 and 75 per cent. solutions of silver nitrate were used.

Procedure.—At the time of bronchoscopy the dog was in a semi-conscious condition and offered little resistance to the procedure. A small amount of mask ether readily quieted the few that were active. A small pledget of cotton attached to a wire rod was saturated with the silver nitrate solution and introduced into a bronchus through a bronchoscope. It was allowed to remain for ten seconds and on removal a whitish eschar remained, the density of which depended upon the strength of the solution employed. The experiments were divided into four groups, the first group being sacrificed at the end of one day; the second at the end of one week; the third at the end of two weeks and the fourth at the end of one month.

As in the earlier experiments, the dogs were quiet, would not eat and appeared somewhat ill for the first two to four days post-operatively. After the first two or three days, a non-productive cough was noted which usually lasted from several days (four to five) to two or three weeks, depending upon the percentage of the solution of silver nitrate used. After the first week, they became quite active and appeared entirely normal except for exhibiting a non-productive cough.

With the expiration of the experiments, the animals were sacrificed and gross and microscopic studies made.

Results.—*Group I (one day).*—(*a*) Five per cent. silver nitrate—one day. At autopsy the lung lobe presented a bluish-red discoloration and induration on the surface opposite the site of injury. The bronchial mucosa was swollen and ulcerated.

Microscopic examination showed necrosis of the bronchial epithelium with some degenerative changes in the musculature. The bronchial wall and surrounding parenchyma exhibited marked œdema with scattered blood pigment in the latter. There was marked infiltration with polymorphonuclear leucocytes and to a much less extent with round cells. (Fig. 1.)

(*b*) Fifteen per cent. silver nitrate—one day. The lung surface presented the same findings of discoloration and induration as seen in I*a*. The bronchial mucosa was swollen, hæmorrhagic and ulcerated.

* This work has been conducted under a grant from the Douglas Smith Foundation for Medical Research of the University of Chicago.

On microscopic examination both bronchial epithelium and musculature were found necrotic. The cartilages and elastic tissue were also partially destroyed. The bronchial lumen contained a purulent exudate. The parenchyma adjacent to the bronchus was œdematous and heavily infiltrated with polymorphonuclears and to a less degree with lymphocytes. The alveolar spaces were entirely obliterated. Scattered blood pigment was much in evidence.

(*c*) Thirty-five per cent. silver nitrate—one day. Grossly the lung surface appeared very much as in I*a and* I*b* except that more tissue was involved. The bronchial mucosa was œdematous and ulcerated, almost completely obliterating the bronchial lumen.

Microscopic examination showed all elements of the bronchial wall to be totally necrotic, with the exception of the cartilages which were only partially destroyed. Much œdema and some hæmorrhage were seen in the surrounding parenchyma with

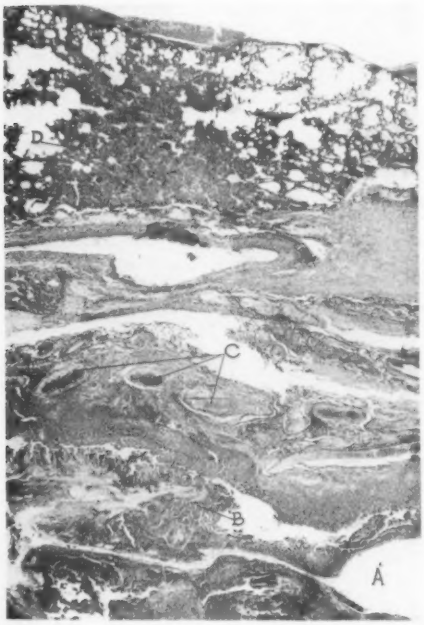

FIG. 1.

FIG. 2.

FIG. 1.—Microscopic appearance of bronchial wall and surrounding parenchyma one day following cauterization with a 5 per cent. solution of silver nitrate. A—Bronchial lumen. B—Infiltrated bronchial mucosa. C—Cartilages little damaged. D—Infiltrated surrounding parenchyma. (x 25.)

FIG. 2.—Microscopic appearance of bronchial wall and surrounding parenchyma one day following cauterization with a 35 per cent. solution of silver nitrate. A—Necrotic bronchial wall. B—Surrounding parenchyma heavily infiltrated. (x 25.)

marked infiltration of neutrophilic leucocytes and round cells. Scattered blood pigment was also much in evidence. (Fig. 2.)

(*d*) Fifty per cent. silver nitrate—one day. The surface of the lung showed much discoloration and induration as described above. The bronchial mucosa was necrotic and the surrounding parenchyma heavily injected.

Examination under the microscope revealed complete necrosis of the entire bronchial wall. The bronchial lumen contained a purulent exudate. The infiltrating process in the surrounding parenchyma was more extensive than seen heretofore. Polymorphonuclear leucocytes and round cells were about equal in number. Much more blood pigment was in evidence with frank hæmorrhage in many places. A few wandering cells filled with blood pigment were present (macrophages). (Fig. 3.)

(*e*) Seventy-five per cent. silver nitrate—one day. The lung surface at the apex of the lobe exhibited a very extensive bluish-red discoloration and induration. An eschar was present at the site of the injury, with much injection of the surrounding parenchyma.

Microscopic examination revealed the bronchial wall and also several millimetres of the surrounding lung parenchyma to be entirely necrotic. The microscopic picture otherwise was very similar to that seen in the 50 per cent. one-day section except that the process was more extensive.

Group II (*one week*).—(*a*) Five per cent. silver nitrate—one week. The surface of the lung lobe appeared grossly normal; however, the bronchus at the site of injury revealed considerable scarring with about 50 per cent. stenosis of its lumen at that point.

Microscopic examination showed the bronchial epithelium partially regenerated. Beneath it were signs of organization evidenced by many small capillaries with forma-

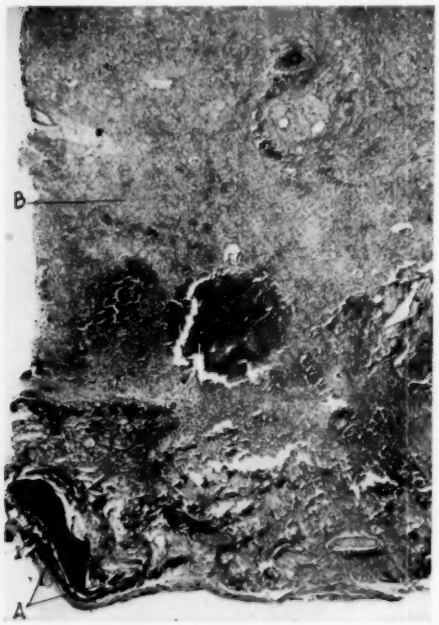

FIG. 3.

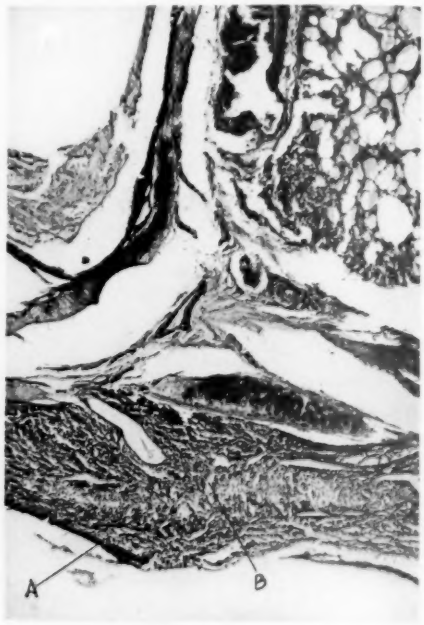

FIG. 4.

FIG. 3.—Microscopic appearance of bronchial wall and surrounding parenchyma one day following cauterization with a 50 per cent. solution of silver nitrate. A—Necrotic bronchial wall. B—Surrounding lung parenchyma heavily infiltrated. (x 25.)

FIG. 4.—Photomicrograph one week following cauterization of bronchus with a 5 per cent. solution of silver nitrate. A—Regenerating bronchial epithelium. B—Connective tissue formation (x 75.)

tion of reticular and fibrillar connective tissue. A considerable amount of blood pigment was present in the reticulum with also a varying amount of granular material. A small number of lymphocytes were still present and fibroblasts were seen everywhere. (Fig. 4.)

(*b*) Fifteen per cent. silver nitrate—one week. The lung lobe was found 100 per cent. atelectatic accompanied by complete stenosis of its primary bronchus.

Microscopic sections revealed partial regeneration of only the bronchial epithelium. A marked round-cell infiltration was still present with many fibroblasts scattered throughout. Very few polymorphonuclear leucocytes were seen. A fair number of plasma-cells had made their appearance. Much evidence of reorganization was seen in the numerous capillaries in the mucosa. Fibrillar connective tissue was present in parts

with scattered blood pigment. The cartilages were extensively damaged, being twisted and misplaced in their relationship. Complete stenosis was accomplished by much fibrillar connective tissue, through which were dispersed the damaged cartilages and lymphocytes. The entire parenchyma was atelectatic, the alveolar walls being in close apposition. The elastic tissue of the bronchus was entirely destroyed. (Figs. 5, 6, and 7.)

(*c*) Thirty-five per cent. silver nitrate—one week. The surface of the lung lobe presented a bluish-red discoloration with moderate induration. The site of injury was located at the division of the primary bronchus into two secondary bronchi. One secondary bronchus was completely stenosed, the other only 50 per cent. stenosed. There was no atelectasis.

Microscopic studies showed much epithelial regeneration. The elastic tissue and bronchial musculature were entirely destroyed. The cartilages were also almost entirely

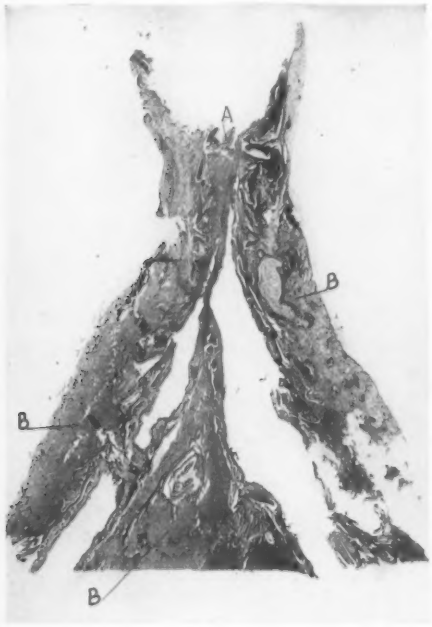

FIG. 5.

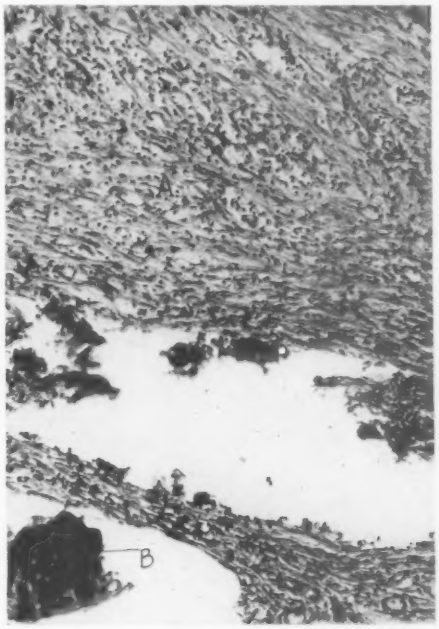

FIG. 6.

FIG. 5.—Microscopic appearance one week following cauterization of bronchus with a 15 per cent. solution of silver nitrate. A—Complete stenosis of bronchial lumen. B and B′—100 per cent. atelectasis of surrounding parenchyma. (x 7.)

Fig. 6.—H.P. at A, Fig. 5. A—Embryonic connective tissue infiltrated with round cells. B—Displaced Cartilage. (x 225.)

necrotic and in some places entirely sequestrated. There was much evidence of organization as shown by the large amount of granulation tissue and fibroblasts. Marked round-cell and polymorphonuclear leucocytic infiltration was still present (about 50 per cent. of each). Much pigment was observed, mostly in phagocytes. Plasma-cells were scattered throughout. The surrounding parenchyma was almost entirely resolved. (Figs. 8 and 9.)

(*d*) Fifty per cent. silver nitrate—one week. The surface of the lung lobe presented the same indurated and injected condition as in the 35 per cent. specimen at this stage, except that it was more extensive. The primary bronchus of the lobe showed complete stenosis and the lung lobe was about 75 per cent. atelectatic.

Microscopic sections revealed partial regeneration of the bronchial epithelium with much granulation-tissue formation. Elastic tissue, muscle and cartilage were entirely

necrotic. The granulations, as well as surrounding parenchyma, were quite heavily infiltrated with round cells and polymorphonuclear leucocytes. Many plasma-cells were present; also phagocytes containing blood pigment. Fibroblastic proliferation was present with reticular connective-tissue formation in some areas. A granular material was seen in the alveoli surrounding the bronchial wall.

(*e*) Seventy-five per cent. silver nitrate—one week. The lung was found to be about 50 per cent. atelectatic, the bronchus being occluded with necrotic material (not stenosed by fibrous tissue formation). Much induration was present at the site of the injury. The entire bronchial wall was in the process of sequestration.

Microscopic studies revealed necrosis of the entire bronchial wall and surrounding parenchyma. The necrotic tissue was in process of sequestration. The bronchial lumen contained pus. A heavy infiltration of lymphocytes and neutrophilic leucocytes entirely obliterated the surrounding viable alveoli.

FIG. 7.—Elastic tissue stain at B of Fig. 5. A—Complete lack of elastic fibres at region of stenosis with derangement of cartilages. B—Elastic fibres of uninjured parts. (x 25.)

More scattered areas of hæmorrhage were apparent than in the preceding one-day stage. Little evidence of regeneration was seen.

Group III (*two weeks*).—(*a*) Five per cent. silver nitrate—two weeks. The lung surface appeared normal. The bronchial lumen revealed scarring with partial stenosis at the site of injury.

Microscopic examination showed complete regeneration of the bronchial epithelium. All evidence of recent inflammation had disappeared, leaving some derangement of the component parts of the bronchial wall as the only evidence of the recent injury.

(*b*) Fifteen per cent. silver nitrate—two weeks. The lung surface appeared normal. The bronchial lumen was scarred and partially stenosed.

Microscopic examination revealed the bronchial epithelium completely regenerated. The bronchial musculature and elastic tissue had been partly destroyed without regeneration. Many of the cartilages had become necrotic, some had disappeared, while others were displaced. Marked round-cell infiltration was still present, with fibroblasts and plasma-cells much in evidence. Much granulation tissue was seen and in parts fibrous-tissue formation. The surrounding parenchyma exhibited some infiltration with lymphocytes with partly phagocytized scattered blood pigment.

(*c*) Thirty-five per cent. silver nitrate—two weeks. The lung lobe was about 15 per cent. atelectatic. Its bronchus was almost completely stenosed, just admitting a two-millimetre probe. The air passages distal to the stenosis contained some collected mucus which was obstructing the air passage.

Microscopic examination revealed partial regeneration of the bronchial epithelium. The bronchial musculature and elastic tissue had been completely destroyed with no signs of regeneration. The cartilages had been severely injured, many were absent, and others displaced. Those remaining at the site of the injury presented much evidence of recent injury. Marked lymphocytic infiltration was still present in some areas. A large amount of fibrous connective tissue appeared to be producing a marked stenosis of the bronchial lumen. One division of the main bronchus was completely stenosed. The surrounding parenchyma presented lymphocytic infiltration with some blood pigment present.

(*d*) Fifty per cent. silver nitrate—two weeks. The lung lobe was 100 per cent. atelectatic with its primary bronchus completely stenosed. The larger air passages were completely plugged distal to the stenosis with retained mucus.

Microscopic studies presented partial regeneration of the bronchial epithelium. The

FIG. 8.

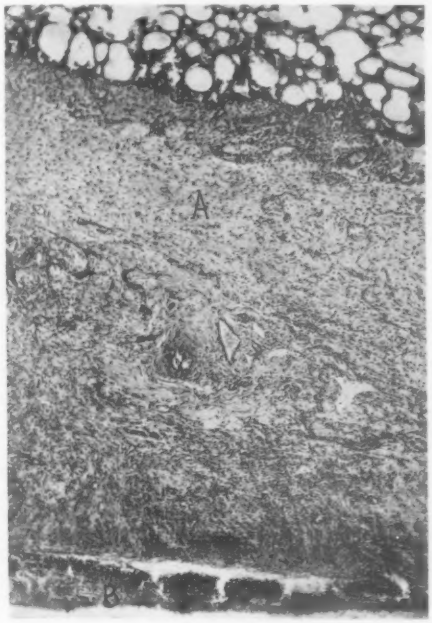

FIG. 9.

FIG. 8.—Microscopic appearance of bronchial wall and surrounding parenchyma one week following cauterization with a 35 per cent. solution of silver nitrate. Note reorganization of A. (x 25.)

FIG. 9.—H.P. at A, Fig. 8. Note reorganization at A near parenchyma with fresh granulations, and infiltrative process at B near surface of bronchial lumen. (x 75.)

bronchial musculature and elastic tissue had been completely destroyed with no evidence of regeneration. The cartilages, as in this stage of the 35 per cent. silver nitrate studies, had been very extensively damaged. Many had been entirely sequestrated; others presented evidence of degeneration and were scattered through the fibrous tissue, producing a stenosis. A variable amount of round-cell infiltration was still present, with marked fibroblastic proliferation and fibrous-tissue formation. The surrounding parenchyma presented a similar picture to that seen in the 35 per cent. study of this stage.

(*e*) Seventy-five per cent. silver nitrate—two weeks. The surface of the lung exhibited much bluish discoloration and induration opposite the site of injury. On section of the lobe, the bronchus was markedly stenosed, with the surrounding tissues swollen and hæmorrhagic. Sequestration of the necrotic bronchial wall was in progress. The air passages distal to the stenosis contained retained mucus.

Microscopic examination presented the entire bronchial wall and also a portion of the surrounding parenchyma entirely necrotic and in the process of sequestration. The bronchial epithelium was partially regenerated at the extremities of the injury. Granulation-tissue formation with a small amount of fibrous tissue was present. There was evidence of lymphocytic infiltration with scattered blood pigment. Extensive hæmorrhage was apparent in the surrounding parenchyma.

Group IV (*one month*).—(*a*) Five per cent. silver nitrate—one month. The lung lobe grossly appeared normal. The bronchus was about 50 per cent. stenosed.

Microscopic examination revealed only a small amount of elastic tissue and bronchial musculature to have been damaged. The cartilages presented some evidence of degeneration, but none was missing. Complete regeneration of the bronchial epithelium had taken place with a tendency to polyp formation. A fair amount of fibroblastic proliferation was in evidence. Most of it was in the formation of connective tissue, as

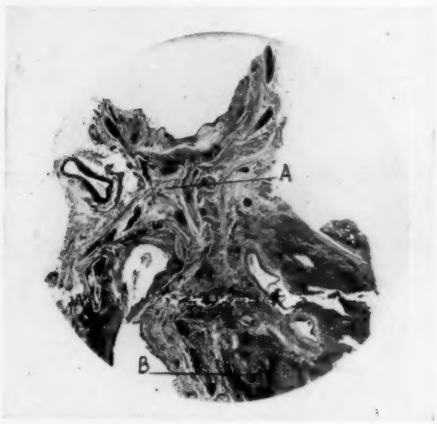

FIG. 10.

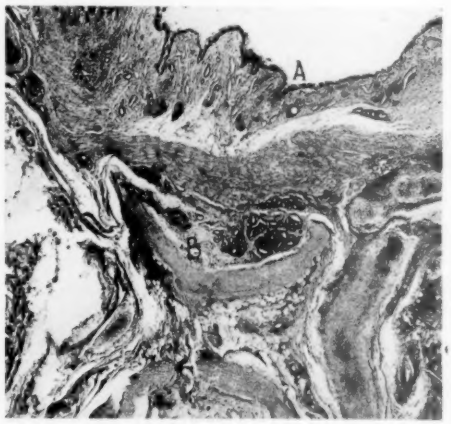

FIG. 11.

FIG. 10.—Photomicrograph one month following cauterization of bronchus with a 50 per cent. solution of silver nitrate. A—Complete stenosis of bronchus. B—100 per cent. atelectasis of surrounding parenchyma. (x 7.)
FIG. 11.—H.P. at A, Fig. 10. Note regenerated bronchial epithelium A, with the dead cartilages intermingled with the fibrous tissue B to produce complete closure of the bronchial lumen. (x 30.)

demonstrated by the small number of capillaries present and a large amount of cellular substance. Very few lymphocytes were seen.

(*b*) Fifteen per cent. silver nitrate—one month. Grossly the findings were very similar to those present in the 5 per cent. study at this stage.

Microscopic examination also revealed changes comparable to those of the former study, with the exception that the cartilages were somewhat displaced and more evidence of injury in elastic and musculature tissues was apparent.

(*c*) Thirty-five per cent. silver nitrate—one month. The lung lobe was 100 per cent. atelectatic. The primary bronchus was completely stenosed with the larger air passages distal to the stenosis plugged with retained mucus.

Microscopic studies revealed partial loss of muscular and elastic tissue. The cartilages were present, but were scattered and showed much evidence of degeneration. The bronchial epithelium had been completely regenerated. Fibrous-tissue formation with intermingling misplaced cartilages produced complete stenosis of the bronchial lumen. Very few lymphocytes were present.

(*d*) Fifty per cent. silver nitrate—one month. The lung lobe grossly presented the same findings as those of the 35 per cent. specimen at this stage.

Microscopic examination revealed partial absence of elastic tissue, bronchial musculature and some of the cartilages. Incomplete regeneration of the bronchial epithelium was observed, with the formation of several inclusion cysts. The stenosis appeared

to be brought about by fibrous-tissue formation with partial collapse of the bronchial wall and by the filling in of the misplaced, partly degenerated cartilages. The surrounding parenchyma was entirely atelectatic. The pulmonary artery lying adjacent to the bronchus revealed destruction of some of its elastic and muscular fibres, with replacement by fibrous connective tissue. (Figs. 10 and 11.)

(*e*) Seventy-five per cent. silver nitrate—one month. The lung surface appeared entirely normal. The primary bronchus was markedly stenosed, apparently in part by granulation tissue.

Microscopic sections exhibited an absence of the entire bronchial wall and a portion of the surrounding parenchyma. The bronchial epithelium was the only constituent seen to be regenerating, and this was incomplete. Much fibrous-tissue formation replaced the destroyed tissue. On the surface of this fibrous tissue, granulations were seen heavily infiltrated by lymphocytes and a fair number of plasma-cells. A good deal of blood pigment was still present, mostly within phagocytes. The surrounding parenchyma was still heavily infiltrated with lymphocytes. A fair amount of œdema and hæmorrhage was also seen, with blood pigment scattered throughout. The pulmonary artery lying next to the bronchus presented more advanced changes but similar to those seen in the 50 per cent. specimens at this stage. (Figs. 12, 13, and 14.)

Comment.—A safe and reliable procedure for closing large bronchi has been presented. This method has been carried out on a large number of dogs (fifty to sixty) with uniform success.

The complete stenosis of a bronchus 0.5 inches in diameter was a routine occurrence within two weeks following the application of a 35 per cent. solution of silver nitrate.[2]

Smaller bronchi (one-quarter inch) became completely stenosed in from one to two weeks following a single application of a 15 per cent. solution. The stenosis in either case was apparently due to partial collapse of the bronchial wall following destruction of its cartilages, with filling of its lumen with granulation tissue which later became fibrous connective tissue. The 50 per cent. solution was also followed by the production of complete stenosis in a variable number of cases (70 per cent.); however, it was found less safe to use (14 per cent. mortality).

This finding was applied in the production of massive atelectasis preliminary to lobectomy and pneumectomy.[3] It also presented a means of closing persistent bronchial fistula produced experimentally.[4]

It has been suggested that it might be used to advantage in closing the primary bronchus of a lobe subsequent to drainage of an abscess from that lobe. This would prevent frequent contamination of the abscess from the tracheo-bronchial tree and might be a valuable aid in clearing up of the disease process, or might act as a preliminary step to cautery pneumectomy or other procedures. Repeated attempts to close the bronchus draining an experimental abscess have been accompanied by failure in each instance.

The value of bronchial stenosis with resultant atelectasis in the treatment of disseminated tuberculosis is under experimental investigation at the present time.

Complete stenosis of the primary bronchus of a lung lobe was accompanied routinely by 100 per cent. atelectasis of the lobe. Complete stenosis

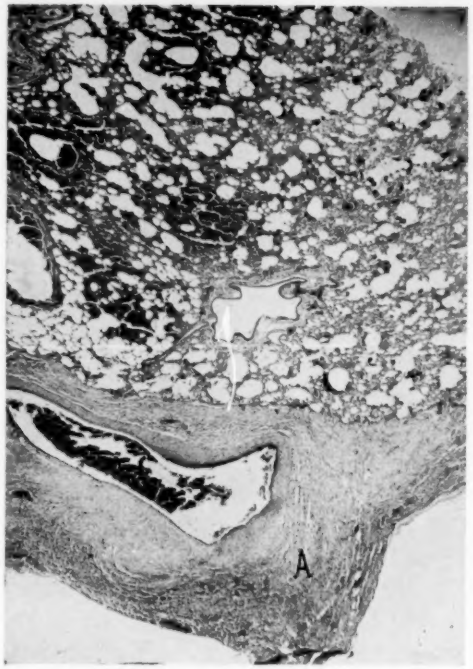

FIG. 12.

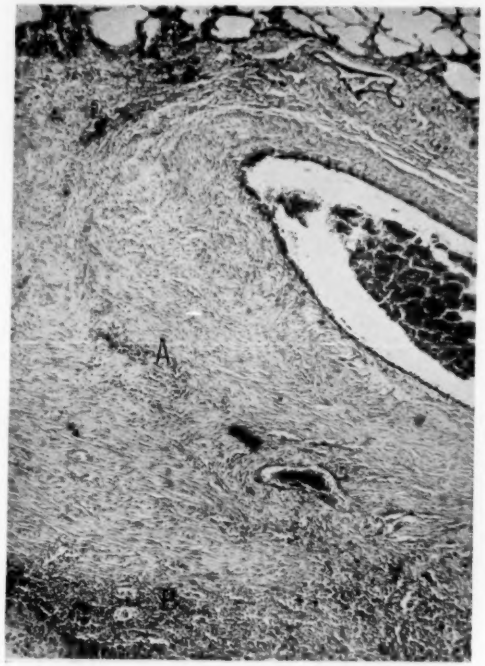

FIG. 13.

FIG. 14.

FIG. 12.—Microscopic appearance one month following cauterization of a bronchus with a 75 per cent. solution of silver nitrate. A—Extreme degree of fibroblastic proliferation (Fig. 13) with some infiltration still present in parenchyma. (x 25.)

FIG. 13.—H.P. at A of Fig. 12. A—Much fibrous tissue formation with some damage to blood-vessel wall. (See Fig. 14.) B—Lymphocytic infiltration still present. (x 75.)

FIG. 14.—H.P. at A of Fig. 12. Elastic tissue stain. Note extensive damage to B.V. wall (A) with loss of elastic tissue and muscular elements and repair by connective tissue formation. (x 75.)

of only one division of the primary bronchus was never accompanied by atelectasis. This method of producing experimental atelectasis was made use of in a study of that condition.

As in the previous studies, necrosis of the entire bronchial wall was followed by regeneration of only the bronchial epithelium. The rapid rate of regeneration was such that an occasional piling-up of epithelial cells was seen with polyp formation. In other areas, epithelial cells were seen beneath the surface forming inclusion cysts. These phenomena were observed by Winternitz[5] in his studies on intrabronchial insufflation of hydrochloric acid in rabbits.

It was of interest to note that damage to the adjacent pulmonary vessels was present following only the higher percentages (50 and 75) of solution. Whereas four out of nine dogs receiving a 75 per cent. solution died of pulmonary hæmorrhage in one to seven days following cauterization, only one out of seven dogs receiving a 50 per cent. solution died of pulmonary hæmorrhage. *No deaths occurred when a lower percentage was used.*

In contra-distinction to the previous study on bronchial injury and repair, no infarcts were produced in the present work. It will be remembered that considerable blood-vessel damage accompanied the use of the silver nitrate "stick," which adequately explains the infarct formation.

Unlike the findings of other workers, no pleural exudate accompanied the production of complete atelectasis in our experiments. The difference in methods of production will perhaps explain this phenomenon, as no local irritation or other unphysiologic process was present at the time collapse was being produced.

CONCLUSIONS

(1) Complete stenosis of a bronchus one-half inch in diameter was the usual occurrence in two weeks following the application of a 35 per cent. solution of silver nitrate (15 and 50 per cent. less often).

(2) Stenosis of the bronchial lumen was brought about by collapse of its wall and by filling in of the lumen with the injured elements of the wall and granulations; with subsequent fibrous-tissue formation.

(3) Massive atelectasis accompanied complete stenosis of the main bronchus of a pulmonary lobe. No atelectasis was present when all of the main air passages to the lobe were not completely obstructed.

(4) The entire wall of a bronchus became necrotic following cauterization with a 75 per cent. solution of silver nitrate. Parts of the wall remained viable when solutions of a lower percentage were used.

(5) The cartilages and elastic tissue were found to be the most resistant to destruction by the solutions employed.

(6) Following necrosis of the entire bronchial wall, only the epithelium was found to regenerate.

(7) Regeneration of the epithelium was very rapid, not infrequently giving rise to polyp and inclusion cyst formation.

(8) Fibroblastic proliferation was very pronounced, especially following 35 and 50 per cent. solutions of the chemical.

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TRAUMATIC RUPTURE OF CONGENITAL HYDRONEPHROTIC KIDNEY

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The so-called congenital hydronephrotic kidney is one type of hydronephrosis usually caused by some form of obstruction at the uretero-pelvic juncture, which develops prior to birth or in early infancy, and in the vast majority of cases manifests itself before the end of the first decade. According to Rovsing, one-third of all cases of hydronephrosis in children are congenital. From a study of 4000 autopsies performed on children, Bugbee found fifty-three cases of this condition and concluded that most of these children die within the first six months of life. Küster reviewed 500 cases and found fifty-one in children under ten years of age. Campbell, who reported 2000 autopsies on children, found that in 2 per cent. of these there was some form of congenital ureteral obstruction and of this number 80 per cent. revealed evidence of infection.

A hydronephrotic kidney in a child often escapes recognition, and the failure of diagnosis is due to the fact that such a kidney is usually unaccompanied by fever or urinary changes. The true nature of the lesion is recognized after a careful X-ray and cystoscopic study.

Hydronephrotic kidneys are more prone to spontaneous and traumatic rupture than normal kidneys. Henline, who made an exhaustive study of cases of spontaneously ruptured kidneys, found only twenty-four cases reported in the literature, nineteen of which were operated upon and of which seven died. Of the five unoperated cases, all died. Pierre Bazy reported one case. Connell reported thirty cases, while Amberger reported one case of spontaneous rupture of hydronephrotic kidneys. LeComte reported one case and Miller reports one case of an intraperitoneal rupture of a hydronephrotic kidney. It is, of course, impossible, after reviewing the reported cases, to rule out with absolute certainty the factor of muscular contraction or some form of mild trauma as etiologic factors in any of them.

Traumatic rupture of the kidney is not as rare as was formerly believed. Küster, from the Clinic at Basle, reports ten cases out of 30,000 admissions, and only 0.12 per cent. of 7741 necropsies; and of all cases dying from trauma, only 8 per cent. were due to renal injuries. Gutterbock, reporting upon 326 necropsies, found 10 per cent. to be due to rupture of the kidney. In contrast with these figures we find today that such an injury is far more frequent, as is, for example, indicated by Delzell and Harrah, who reported a series of eleven cases.

Renal injuries, as evidenced in the literature, are far more frequent in adults than in children. It is also of interest to note that the right kidney

is more frequently involved than the left. Shapiro reports five cases, all of which were on the right side. Gibson reported four cases in children between the ages of eight and twelve, and stated that up to that time only twenty-two cases had been reported. In none of these cases were the kidneys noted as being hydronephrotic. In all of Gibson's cases the lesions were exactly alike in that the kidney was completely divided into two parts, the line of rupture running vertical to the long axis at the junction of the lower two-thirds, and he explains this on the theory of bursting by hydraulic pressure.

Etiology:—The relative rarity of this lesion in children is due to the lack of exposure of children to the various types of trauma commonly encountered by adult males. The weight of opinion seems to favor Kuttner's theory that as the kidney is a semifluid body, it will tend to burst, under favorable circumstances, along the line of least resistance, following the law of hydraulics. At times, the extent of the renal damage bears no relationship to the severity of the trauma, as is so well shown in two of Gibson's cases, where the severest type of renal damage was unaccompanied by any marks of external violence on the surface of the body.

In children, as in adults, rupture may occur from direct violence during which the kidney is thrown against the lower ribs or against the transverse processes of the upper two lumbar vertebræ, or from indirect violence as in the case of a person who lands upon his feet after a fall, from sudden muscular contraction or spontaneously. The latter, although very interesting and rather unusual as previously outlined, will not bear too careful scrutiny, since it is well nigh impossible to rule out the element of muscular contraction or mild forms of trauma so frequently overlooked by patients.

Pathology.—A trauma of the kidney may, as indicated by Bugbee, result in (*a*) slight laceration of the fatty capsule, (*b*) subcapsular hæmorrhage, (*c*) contusion or laceration of the parenchyma, (*d*) complete laceration or pulpification of the kidney, (*e*) complete or incomplete laceration of the pelvis, ureter and pedicle, (*f*) tear of the peritoneum, or (*g*) an injury to other viscera. Contrary to expectation, the extent of the renal damage does not always depend upon the severity of the trauma producing it. At times a severe trauma will only produce a subcapsular rupture which will subside in a few days and give no further trouble; and at other times a mild injury will result in a complete rupture with laceration of the renal pedicle and lead to exsanguination or to a penetration of the peritoneum and a fatal peritonitis.

A subcapsular renal rupture will, if permitted to go untreated, lead to an accumulation of blood under the capsule, which will eventually result in an automatic tamponade of the kidney and a cessation of the hæmorrhage, and finally to an absorption of the extravasation, connective tissue replacement of the destroyed renal parenchymal elements and to spontaneous recovery.

Where the rupture is complete, that is, where the tear is through the capsule, blood and urine escape into the perirenal tissues and form a retroperitoneal collection of considerable size, which can often be felt as an elastic

tumor. In those cases where the peritoneum is capable of withstanding the increasing pressure of the extravasated fluid, the pressure will eventually reach a point where it will equalize that existing within the bleeding vessel and the hæmorrhage automatically ceases. If such a case is permitted to go untreated, one of two things may happen. In favorable cases the exudate is absorbed and the patient recovers. On the other hand, should infection supervene, the patient may succumb to general sepsis, or, by direct extension, to a fatal peritonitis. When the rupture includes the peritoneum, the patient may die of hæmorrhage or peritonitis.

Although most traumatized kidneys lead to hæmaturia, this is not an invariable finding since it is possible for some of the collecting tubules in the renal parenchyma to become occluded by clots and thus prevent blood from entering the pelvis and ureter. Similarly, if there is a complete severance of the pelvis from the ureter, it is obvious that blood will be unable to reach the bladder. Where a communication exists between the kidney and peritoneal cavity, bleeding will take place there and not in the bladder. It is for these reasons that Vielcker claims that hæmaturia is not a constant symptom in rupture of the kidney.

The finding of a small spot of ecchymosis may at times lead to the suspicion of a traumatic lesion involving the kidney. The ecchymosis may be situated in the skin over the lumbar region, or, due to burrowing of the extravasation, along the fascial planes, may be found in the thigh, over the base of the penis, scrotum or in the perineum, as was seen in one of the cases reported by Delzell and Harrah.

Symptoms.—The three outstanding symptoms associated with rupture of the kidney are pain, hæmorrhage and tumor. Pain may be mild or severe, and at times assume the character of renal colic radiating to the thigh or bladder. Fieschi reported a case where the pain was on the opposite side to the side of the lesion. The intensity of the pain bears no relationship to the severity of the renal lesion, since kidneys almost completely pulpified may be accompanied by little pain. This phenomenon is explained by the fact that the renal parenchyma as shown by Papin is devoid of sensory nerves, so that any pain which is experienced is due to tension produced by the extravasation or to the passage of clots down the ureter.

Although the presence of a swelling in the loin is evident in most cases, it is absent where the renal laceration is associated with a rent in the peritoneum. The swelling which is soft and fluctuant is situated in the loin, may be small or large, and is due to an extravasation between the kidney and the peritoneum. The presence of such a tumefaction is often obscured by the rigidity of the overlying abdominal and lumbar muscles. Hæmaturia which is present in most though not all of the collected cases may be constant or intermittent. It may be profuse or scanty and the extent of the hæmaturia is at times out of all proportions to the severity of the lesion. Shock is a variable feature. When present it may be due to the renal injury, to hæmorrhage, peritoneal irritation or to associated injuries to other organs. Fever

accompanies infection and is often present in the later stages during the absorption of the extravasation.

A history of a pre-existing enlargement of a kidney in a child suggestive of hydronephrosis will often give a valuable clue to the existence of a laceration in cases of spontaneous rupture or rupture following some trivial trauma.

Diagnosis.—A tentative pre-operative diagnosis of rupture is possible in cases where a history of trauma is elicited along with the three outstanding symptoms, namely, pain, hæmaturia and a fluctuating mass in the loin. An absolute diagnosis can be made if, along with the foregoing features, positive pyelographic data are obtainable, such as the finding of the dye outside the renal silhouette or distributed through the renal parenchyma.

Treatment.—The treatment of rupture of the kidney falls into three groups: (*a*) conservative non-operative or expectant, (*b*) conservative operative, (*c*) nephrectomy. The type of treatment must of necessity depend upon the existing pathology. Cases of laceration of the fatty capsule, or mild contusions or subcapsular lacerations of the parenchyma call for nothing more than a few day's rest in bed, and the application of ice-bags to the traumatized side. Surgical intervention is indicated only when the patient, who is placed upon careful expectant treatment, continues to show signs of progressive hæmorrhage or shock. In such cases the employment of conservative or radical surgery depends entirely upon the lesion present in the exposed kidney. Certain lacerations, especially those in which the rent has not involved the vascular pedicle of the kidney, can be well treated by packing or suture, or by a combination of both, associated with ample drainage. Nephrectomy must be performed upon those cases where the kidney is hopelessly pulpified, the pedicle torn or the pelvis severed from the ureter, or in those cases where the injury has occurred in congenital hydronephrosis or in other hopelessly diseased kidneys.

In the event of peritoneal damage, laparotomy must be performed, and it is here where the ingenuity of the surgeon is taxed to the utmost, especially in those cases where doubt exists as to whether the lesion is entirely extra or both extra and intraperitoneal. It is the writer's belief that, in those cases where it is impossible to ascertain the presence of intraperitoneal leakage or damage in addition to the renal injury, it is best to perform a laparotomy for the purpose of thorough exploration and the establishment of appropriate drainage if necessary, before exposing the kidney through the lumbar route. It is not considered good practice in this type of case to explore the kidney transperitoneally. A much safer procedure is to drain the retroperitoneal extravasation through the lumbar route. In cases where doubt exists regarding the advisability of saving the kidney, it may be best to drain the exudate at the first operation and, if nephrectomy becomes necessary, to perform it at a subsequent date when the patient's condition has improved and the risks of infection reduced. This is especially true in cases of rupture of a congenital hydronephrotic kidney where at operation the kidney has collapsed into a small sac, making nephrectomy difficult and hazardous.

CASE REPORTS.—CASE I—M. G., male child, aged six years, was first seen September 3, 1930, complaining of pain in the right lower quadrant of the abdomen and vomiting of two days' duration. Three years previously the abdomen was explored for suspected appendicitis. A normal appendix was removed but the operating surgeon reported finding a polycystic kidney on the right side, no comment being made on the condition of the left kidney. Three weeks ago, he suffered a convulsion.

Two days prior to present consultation, while playing, he struck his abdomen against an iron bar. Immediately after the accident he complained of pain in the right side of the abdomen and vomited. The pain in the abdomen and vomiting continued and the temperature rose to 102°. There was no hæmaturia. Examination of the

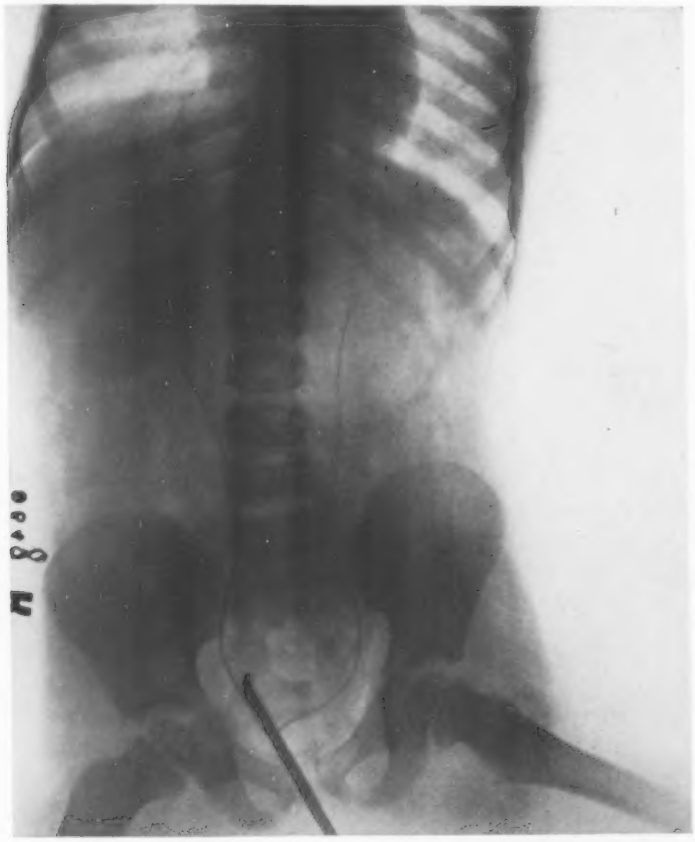

FIG. 1.—Case I. Right pyelogram showing obliteration of normal landmarks of pelvis and calices.

abdomen revealed diffuse tenderness, most marked over the right iliac fossa and right loin, and rigidity of the abdominal musculature, especially the right rectus with a positive rebound. The muscles over the right lumbar region were also spastic. Temperature, 101°. Pulse, 120. Blood-count showed 30,000 leucocytes per cubic millimetres of which there were 60 per cent. segmented neutrophiles and 14 per cent. band-forms, 14 per cent. small lymphocytes and 2 per cent. monocytes. Urinalysis showed a faint trace of albumin, a few leucocytes, an occasional hyaline cast and 2 plus urobilin. There were no red cells.

From the history and physical signs, it was impossible to rule out an intra-abdominal lesion. The history that another surgeon had found a polycystic kidney on the right side in the course of an abdominal exploration lead to a strong conviction that we

were dealing with a primary lesion involving the right kidney. It was decided first to rule out any intra-abdominal pathology by performing a preliminary laparotomy to be followed immediately by an exploration of the kidney through the usual lumbar route.

Operation.—September 2, 1930. Under general anæsthesia the abdomen was opened through a three-and-a-half inch right rectus muscle-splitting incision. Situated behind the ascending colon and lifting it away from the posterior abdominal wall there was a large retroperitoneal, dark mahogany-colored extravasation. There was no evidence of leakage into the peritoneal cavity. The abdomen was quickly closed in layers and the patient turned on the left side, and through a three-inch Albarran incision the right renal fossa was exposed, revealing a large collection of blood and urine. This was removed by suction. In place of a palpable renal mass, a small collapsed sac was felt which appeared to be the remains of the kidney. In view of the condition of the patient, and lack of information concerning the exact state of the other kidney, it was thought advisable to drain the wound and terminate the operation at this time. Two rubber tubes and one dam were introduced and the wound closed in layers.

FIG. 2.—Case I. Photograph taken of kidney immediately after removal.

FIG. 3.—Case I. Kidney opened showing complete atrophy of renal parenchyma.

Blood chemistry, September 3, urea n., 13.5; creatinin, 1.6; glucose, 79. September 18, urea n., 6.7; creatinin, 1.2; glucose, 77.

Both wounds were healed on September 22, 1930, and the patient was permitted out of bed. On September 26, uroselectan was given intravenously. The röntgenograms revealed a failure of the right kidney to take the dye. Although the left renal pelvis was somewhat dilated, the pyelogram failed to reveal any features suggestive of polycystic disease. Repeated urinalyses were negative. Patient was discharged September 27.

On December 13, 1930, the child was seen again. Since his discharge from the hospital he had gained weight and felt well. He voided six times during the day and did not have to void during the night. At 6 A. M. on the day of the present examination the patient voided bloody urine. The mother stated that four days previously, while playing, he again struck his right flank against a chair.

Cystoscopy revealed many red blood-cells and clumps of pus-cells in the urine from the right kidney while that from the left was negative. Bilateral shadowgraphy was negative. Right pyelography showed a completely destroyed hydronephrotic kidney. (Fig. 1.)

Operation.—December 17, 1930. The right kidney was exposed through a four inch Albarran incision. The kidney was enormously enlarged and converted into a thin-walled sac. The pelvis was also enormously dilated and the ureter appeared to be implanted into the posterior wall of the pelvis, so that it formed an acute angulation upwards and backwards from the most depended portion of the pelvis. The uretero-

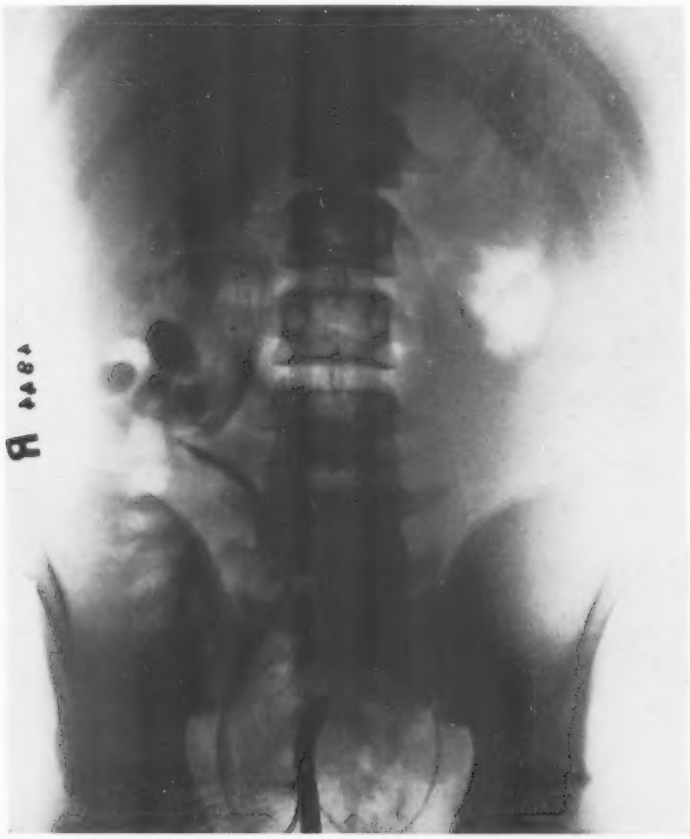

FIG. 4.—Case II. Right pyelogram showing irregular distribution of the dye through the renal silhouette.

pelvic opening was very minute. The kidney was easily removed and the wound closed in layers after draining the renal fossa.

Pathological Report.—Specimen is a kidney measuring 12 by 8½ by 5 centimetres. The surface is bluish-gray, and presents many fibrous adhesions. The kidney is converted into a fluid sac with a thin parchment-like wall. The pelvis is much dilated and the uretero-pelvic orifice, which is situated on the posterior pelvic wall, is very small. On section, the kidney appears like a thin-walled sac containing several loculi lined with a smooth, light gray membrane. The cortex is from two to three millimetres in thickness and contains very little recognizable renal tissue. Sections made through the sac wall show some remains of renal tissue with much fibrosis and inflammatory exudate. *Diagnosis.*—Complete hydronephrosis. (Figs. 2 and 3.)

Following an uneventful convalescence, patient was discharged from the hospital January 5, 1931.

Comment.—This case deals with a boy six years of age who, following an injury to the abdominal wall, sustained a rupture of a congenital hydronephrotic kidney. In spite of the suspicion entertained pre-operatively regarding the nature of the injury, the signs and symptoms pointed so strongly to an intraperitoneal lesion that laparotomy had to be performed in order to rule out such a lesion before exposing the kidney through a lumbar incision. Exploration of the renal region revealed a collapsed, ruptured, hydronephrotic sac with a large retroperitoneal extravasation which was drained at the first operation, and followed by nephrectomy at a subsequent date. It is noteworthy that at no time during the first episode was hæmaturia noted. This symptom occurred prior to the second operation, at which time the kidney, although supposedly traumatized, was not ruptured.

CASE II—A. G., aged fifteen, was first seen January 16, 1928, complaining of hæma-

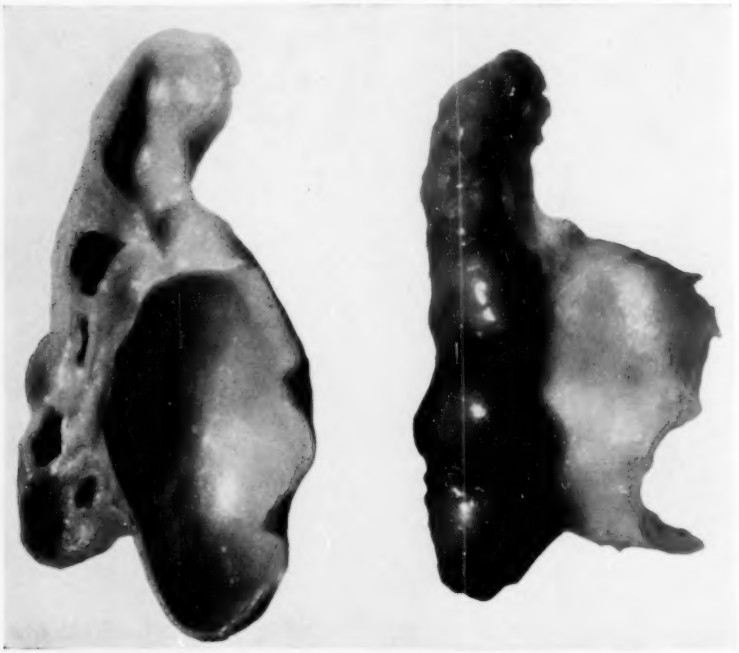

FIG. 5.—Case II. Photograph of kidney showing dilation of pelvis, atrophy of renal parenchyma, and rent in pelvis.

turia of ten days' duration. Past history was negative save for a dull pain in the right flank on and off for several years.

While playing basketball ten days previously, he was struck a light glancing blow on the abdomen, following which he passed a small amount of bloody urine. Hæmaturia persisted for twelve hours, following which he was without symptoms for one week. Three days previously he was again struck a light blow on the abdomen while playing basketball following which he collapsed. A physician who saw him immediately after the accident found him in shock, from which he quickly emerged after mild stimulation. Since then his urine has been bloody. There was no frequency, dysuria or backache. Temperature, 100°. Pulse, 80 per minute.

Cystoscopy revealed a normal bladder. Both kidneys were easily catheterized. There was no flow from the right catheter. Urine from the left kidney contained an occasional red blood-cell and showed good dye concentration.

On the röntgenogram the tip of the opaque catheter was seen to impinge at a point one-half inch above the iliac crest. Right pyelogram showed a complete distortion of the pelvic contour with the dye scattered throughout the renal fossa. The left renal silhouette appeared normal. (Fig. 4.)

Physical examination was essentially negative save for right costovertebral tenderness and spasticity of the lumbar musculature. A pre-operative diagnosis of ruptured kidney was made and operation advised.

Operation.—The right kidney was exposed through a six-inch Albarran incision. The kidney felt soft and boggy and consisted mostly of a large dilated pelvis with very little renal parenchyma. On the ventral surface of the pelvis there was a rent about one inch in length. The entire pelvis was filled with blood clots and there was a large blood-stained extravasation in the perirenal tissues and retroperitoneal space extending down behind the ascending colon. The renal pedicle appeared to enter the kidney near its upper pole. (Fig. 5.)

The kidney was removed and the perirenal space drained. Following a stormy convalescence with temperature ranging between 99° and 103°, the patient was discharged well on January 31, 1928—fifteen days after operation.

Comment.—This case deals with a boy fifteen years of age who sustained a rupture of a hydronephrotic kidney undoubtedly congenital in origin, following a slight trauma during a basketball game. Although hæmaturia was noted following the accident, it persisted for only twelve hours and apparently resulted in little discomfort. One week later, following a second injury, he collapsed and at operation a ruptured hydronephrotic kidney was removed. The probabilities are that during the first trauma the kidney sustained only a subcapsule laceration which perforated after the second trauma, resulting in a retroperitoneal extravasation.

SUMMARY AND CONCLUSIONS

(1) The incidence of rupture of congenital hydronephrotic kidneys is small.

(2) Kidneys in children which rupture as a result of mild traumata are usually hydronephrotic.

(3) So-called spontaneous rupture of kidneys may be due to mild forms of trauma easily overlooked by patients. They are very rare, as attested to by a paucity of cases reported in the literature.

(4) Hæmaturia is not a cardinal symptom in rupture of the kidney.

(5) The presence of ecchymotic areas over the skin of the loin, thigh, penis or perineum may be suggestive of a renal injury.

(6) Exploratory laparotomy is indicated in all cases where doubt exists regarding the presence of an intra-abdominal lesion associated with the renal injury. The kidney should be exposed and explored through a second incision in the loin.

(7) Retroperitoneal extravasations are best drained through the lumbar route rather than transperitoneally.

(8) Conservative surgery cannot be employed in cases of ruptured congenital hydronephrotic kidneys, nephrectomy being the procedure of choice.

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CYST OF THE PANCREAS ASSOCIATED WITH ECTOPIC SPLENIC ISLAND

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The first successful removal of a pancreatic cyst was reported in 1881 by Bozeman[1] before the New York Pathological Society. The next year, Gussenbauer devised the operation of marsupialization, which is still the commonest method of surgical treatment. Senn[2] reported a case successfully treated by surgery in 1885 and attempted to produce cysts experimentally by tying off the pancreatic duct. Lazarus[3] produced a cyst in the pancreas of a dog. He crushed the pancreas and caused the formation of a hæmatoma about the size of a pigeon's egg, which, after forty days, was converted into a cyst with a smooth, fibrous capsule containing 100 cubic centimetres of a watery fluid. Others attempted to produce cysts experimentally by injecting various substances into the duct of Wirsung. Opie[4] called attention to the relationship between diseases of the pancreas and obstruction to the normal outflow of bile by a stone in the common bile-duct. Eha[5] has reported a cyst the size of an orange in an infant five months old. He believed it to be a congenital cyst. Railton[6] and Shattuck[6] report similar cases in infants. Robson and Moynihan[7] refer to three cases and state that congenital cystic disease of the pancreas is exceedingly rare. The case I wish to report is especially interesting from the standpoint of etiology because of a congenital anomaly found in the pancreas post-mortem.

Case Report.—E. K., a cabinetmaker, sixty-one years of age, was admitted to the Albany Hospital December 9, 1929, into the service of Dr. A. H. Traver. His past history and family history were negative. He had always considered his health good, and his habits were temperate. Five weeks before admission he had severe pain across the upper part of his abdomen and vomited persistently for three days. He attributed this upset to some sardines that he had eaten. There was no jaundice. He had one chill but thought he had had no fever. Following this attack he had no more pain, but his appetite failed, and he felt weak. He noticed that his abdomen began slowly to increase in size. At the time of admission to the hospital his temperature and pulse were normal. He complained only of weakness and the swelling of his abdomen. There was no history of injury.

The physical examination was essentially negative except for a mass filling the upper half of the abdomen. This was smooth and was dull to percussion. No fluid wave or shifting dullness in the flanks could be made out. No definite edge could be felt. There was no apparent enlargement of the liver. The mass was not movable. There was no pulsation or bruit. A twenty-four-hour specimen of urine was negative for sugar, and each of four single specimens collected before and after operation were negative for sugar. The tests for albumen, acetone, and diacetic acid were negative. Examination of the blood showed 4,500,000 red cells and 8,600 white cells, with a normal differential. Hæmoglobin was 70 per cent. by the Tallquist scale. The blood Wassermann

was negative. Fasting blood sugar was 112 milligrams and the non-protein nitrogen was 54 milligrams per 100 cubic centimetres. X-ray examination after a barium meal showed no constrictions or filling defects. The transverse colon was displaced downward by a large rounded mass above it. The greater curvature of the stomach was indented by this same mass. The X-ray findings suggested a large mass outside the gastro-intestinal tract, probably a retroperitoneal tumor.

The pre-operative diagnosis of pancreatic cyst was based on the location of the tumor and its steady increase in size over a period of five weeks after an attack of upper abdominal pain, whose severity and radiation from right to left suggested an acute pancreatitis. In making a diagnosis, the following possibilities were considered: Retroperitoneal cyst or cyst of the mesentery, echinococcus cyst, splenomegaly, aneurism of the abdominal aorta, and ascites. Hydronephrosis or pyonephrosis seemed to be ruled out by the examination of the urine and the fact that the mass did not extend into the flank so as to fill the costovertebral angle when palpated bimanually.

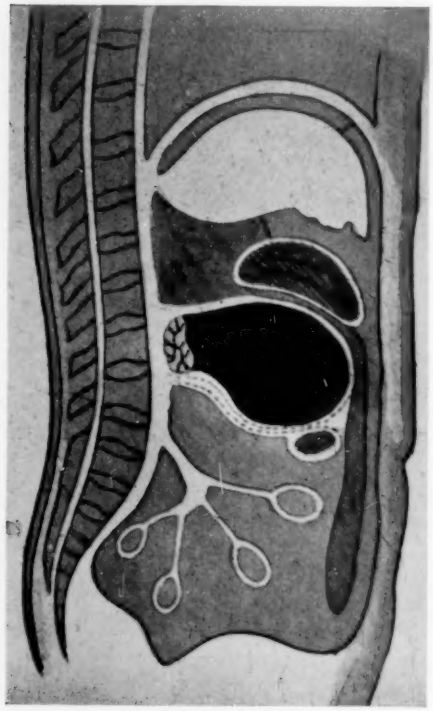

FIG. 1.—Cyst projecting forward from pancreas and presenting between stomach and colon. (From Robson and Cammidge.)

On December 11, operation was performed by Dr. A. H. Traver under ether anæsthesia. When the peritoneum was opened through an upper left rectus incision, a cystic swelling presented itself at once. It was the size of a football and protruded between the stomach and the transverse colon. There were adhesions about the gall-bladder. Palpation revealed that the cyst was separate from stomach, kidney, spleen, and liver. It was approached through the lesser peritoneal cavity by making an opening in the mesocolon. No fat necrosis was seen. The cyst was thin-walled, and fluid leaked out when an attempt was made to put in purse-string sutures, so gauze was packed about the cyst and an Ochsner's trocar was introduced. About a quart of thin, muddy fluid, which looked as if it contained old blood, was drawn into a basin; and then it was possible to enlarge the opening in the cyst and stitch it to the edge of the parietal peritoneum (*i.e.*, marsupialization). A large tube was fastened in place to drain the cyst, and fifteen ounces of fluid were collected in the first twenty-four hours. A stab wound was made at a lower point in the abdomen, and a "cigarette" wick was inserted before closing the primary incision. The fluid from the cyst was examined by Professor Arthur Knudson for amylase by the method of T. R. Brown,[8] using starch and iodine to determine the enzyme action. Only 0.8 per cent. of the amount in normal pancreatic juice was found.

The immediate post-operative recovery was good. There was profuse drainage for a week. After four or five days the tube became loose and fluid escaped around the tube in sufficient quantities to soak the dressings, the binder, and the bed linen. The skin was protected by zinc oxide ointment to prevent digestion about the wound. The patient was coöperative and took large quantities of liquids. Normal saline was given by rectum and by hypodermoclysis. When the temperature rose on the fifteenth day after operation, the wound was irrigated, and chunks of digested tissue, a cheesy material, were washed out. A week later, twenty-one days after operation, the patient died of pulmonary embolism.

Necropsy Report.—An autopsy was performed by Dr. Victor C. Jacobsen only thirty minutes after death, which was fortunate inasmuch as there are very rapid changes in the pancreas post-mortem. The important findings were: A left rectus operative incision 16 centimetres long, 4 centimetres to the left of the umbilicus. From the upper end of this wound a purulent material exuded, and the wound was partly open. To the inside of the wound the omentum was adherent, and small white areas of fat necrosis were scattered in the omentum, the anterior abdominal wall, the mesentery, and about the upper part of the abdominal cavity. There were dense adhesions about the spleen, the appendix, and the gall-bladder. Anterior to and slightly below the pancreas was a large cavity lined by an injected membrane. The cavity extended the entire length of the pancreas; it had dissected down to the pole of the left kidney, included the entire lesser peritoneal cavity, and extended into the omentum. In the lumen of the cavity was

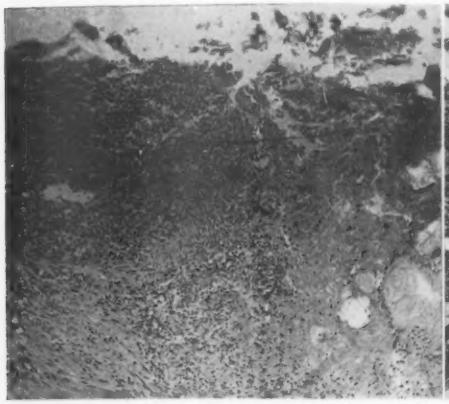

FIG. 2.—Wall of pancreatic cyst. To the left are much fibrosis and lymphocytic infiltration.

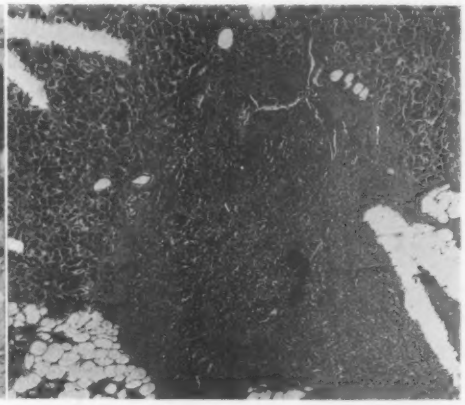

FIG. 3.—A section of the pancreas. In the central portion is an island of splenic pulp in the capsule of which are several dilated pancreatic ducts.

a grayish, caseous, soft substance, resembling necrotic fat. A sinus connected this cavity with the abdominal wound described above. The entire mass was removed and cross-sections made to show, if possible, any connection between the cavity and the pancreatic ducts, but none could be demonstrated. The larger pancreatic ducts were dilated but probing revealed no obstruction. There was considerable fat necrosis throughout the entire gland. Microscopic examination of the pancreas shows much chronic interacinar and interlobular inflammation; also dilatation of some ducts, but many are quite normal. There is hyperplasia of the lining epithelium of some ducts. In one section there is *an ectopic focus of splenic pulp entirely surrounded by pancreatic tissue.* The islands of Langerhans appear normal. In the interlobar fat and capsular fat are large areas of necrosis with moderate inflammatory reaction, but no hæmorrhage. There are cholesterin and hæmatoidin crystals and calcareous salts in some of these areas. The large tract found leading in various directions from the pancreas and down into the mesentery is lined by necrotic tissue and degenerated fat, but no definite epithelial lining is found except a few cells in one section. The conformation of the tract and the finding of ectopic splenic tissue in the pancreas suggest the possibility of some congenital anomaly of pancreatic ducts with cyst formation and subsequent rupture of the cyst, the contents dissecting in various directions and liberating fat-splitting enzymes.

The gall-bladder was markedly dilated, the bladder wall slightly thickened, and the mucosa covered with cholesterol deposits. In the lumen of the gall-bladder were about ten small cholesterol stones. The bile-ducts were markedly dilated, but patent. The right lung weighed 340 grams. There was thrombosis of nearly all branches of the pulmonary artery.

Discussion.—Pancreatic cysts are rather rare inasmuch as White[9] found only three cases in 6,078 autopsies performed at Guy's Hospital in London. They have been classified by Robson and Cammidge[10] as follows:

(1) Retention cysts, which are lined with epithelium and are caused by obstruction in the pancreatic duct, smaller ducts or acini.

(2) Proliferation cysts, due to a proliferation of glandular epithelium followed by an accumulation of fluid. These are true tumors (cystadenoma or cystic epithelioma).

(3) Congenital cysts, analogous to those found in liver, spleen, or kidneys.

(4) Hæmorrhagic cysts, due to hæmorrhagic necrosis.

(5) Hydatid cysts.

(6) Pseudocysts, produced by trauma or degenerative changes of the interstitial tissue of the pancreas. They are distinguished from true cysts in that they are not within the substance of the pancreas but are usually in the lesser omental sac.

(7) Dermoid cysts.

Contents and Location of the Cyst.—The contents of pancreatic cysts vary. The fluid is ordinarily alkaline and has a specific gravity of 1.010 to 1.020. The fluid may be perfectly clear, though usually blood is present, often having undergone marked changes to a dark chocolate or coffee-ground appearance. In the case presented above, there was evidence of old hæmorrhage. The fluid frequently contains one or more of the pancreatic ferments, and it is possible that all three may be present. However, an absence of the ferments does not in any way indicate that a cyst is not of pancreatic origin, for the ferments frequently disappear in the old cysts, often reappearing in the discharge when the cyst is drained.[11] The contents of the pseudocyst are produced by liquefaction of necrotic tissue together with a bloody and inflammatory exudate.

The cysts are frequently located between the stomach and the transverse colon or above the stomach, and less frequently between the layers of the mesocolon. However, they may occupy any part of the abdominal cavity and frequently simulate ovarian cysts.

Etiology.—The pancreas crosses the body of the first lumbar vertebra and may be injured when there is compression of the abdomen, particularly if the abdominal muscles are relaxed and the stomach is empty at the time of the accident.[12] Injury to the pancreas is frequently overlooked when there is an associated injury to other abdominal viscera. According to Heiberg,[13] in one-third of all cases of pancreatic cysts trauma was the cause of the trouble. The enlargement usually appears at once, but it may occur months later, according to Honigman.[14]

Senn classifies the causes of retention as follows:

(1) Obstruction to the outflow of the secretion from impaction of pancreatic calculi in the pancreatic duct or of biliary calculus in the ampulla of Vater.

(2) Partial or complete obliteration of a portion of the pancreatic duct from cicatricial contraction.

(3) Sudden or gradual obstruction of the duct without diminution of its lumen from displacements of the pancreas. Such a displacement may be the result of relaxation of the attachments to surrounding structures, to pressure on the gland from tumors or exudation, or to cicatricial contraction in the substance of the gland.

Hæmorrhage seems to be an important etiologic factor in many cases (Warnock,[15] Lloyd[16]), as a hæmatoma has been found in the substance of the pancreas during exploration of a pancreatic cyst. Many of the pseudocysts, according to Lloyd, are fluid effusions into the lesser peritoneal sac, the result of injury to the underlying pancreas, and not cysts of the pancreas in the proper meaning of the term. Other cases seem to follow an acute pancreatitis, which in turn may be due to a plugging of the ampulla of Vater with a gall-stone and the back flow of bile into the pancreas.[4] Experimentally, however, Mann and Giordano[17] have found it impossible to produce a pancreatitis by such means unless bile is injected directly into the duct of Wirsung under considerable pressure, and then they assume the inflammation and subsequent cyst formation are due to rupturing of some of the small ducts and an escape of pancreatic fluid rather than bile alone.

Diagnosis.—The diagnosis can be made only when the cyst has attained considerable size. Pain is not a constant symptom unless it comes from pressure or from associated conditions. In non-traumatic cases the history may suggest an acute or chronic pancreatitis or a biliary colic preceding the development of the cystic tumor. Frequently, the patient complains only of the increasing swelling of the abdomen with loss of appetite. There may be large fatty stools, and there may be sugar in the urine, but these findings are often absent if the pancreatic function is maintained.

Physical examination reveals a rounded, firm, smooth tumor of varying size in the epigastrium. It moves with respiration and is separate from liver, spleen, or kidney. The tumor may be fluctuant and may transmit the pulsations of the abdominal aorta. It is not usually tender. The stomach can usually be made out by percussion lying above the tumor and to the left, and sometimes the transverse colon can be made out passing anterior to it. When the cyst fills the abdomen, there may be a fluid wave, but there should be no shifting dullness in the flanks. A rectal or vaginal examination should help to distinguish between pancreatic cyst and ovarian cyst.

Treatment.—The treatment of these cases is always surgical and is most often simple drainage after drawing the cyst up into the wound and stitching it to the parietal peritoneum at the edge of the incision (*i.e.*, marsupialization). In some cases the operation has been done in two stages, but this is not often necessary. Aspiration by means of a trocar or needle inserted through the intact abdominal wall is no longer considered good surgery as the stomach or transverse colon may be compressed and intervene. Excision of a part or the whole of the cyst would seem advisable if this were possible

without producing an extensive hæmorrhage. Prolonged drainage is indicated.[18] The mortality of such operations is from 20 to 30 per cent. A few patients require a secondary operation because the cyst refills or because of malignant degeneration. Occasionally, the sinus formed after an operation refuses to heal; and when a total loss of pancreatic secretion occurs, this is a serious matter producing dehydration, emaciation, extreme weakness, a diarrhœa with large fatty stools, and finally death. Radium has been used with good results in a few cases to promote fibrosis and healing when there is a persistent sinus.[19]

SUMMARY

(1) A case of cyst of the pancreas is reported in a man of sixty-one years, who died twenty-one days after operation, of pulmonary embolism.

(2) The cyst had ruptured, the contents causing widespread fat necrosis. The lining of the cyst suggested an origin from pancreatic ducts, with possibly a congenital anomaly of ducts as a basis for the condition. This hypothesis is supported by the presence of another anomaly, splenic tissue within the pancreas.

(3) Other theories of etiology are discussed.

(4) Treatment consists of early operation with prolonged drainage. Complications are frequent.

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TRANSACTIONS

OF THE

NEW YORK SURGICAL SOCIETY

STATED MEETING HELD APRIL 22, 1931

The President, Dr. Edwin Beer, in the Chair

JEJUNO-COLIC FISTULA FOLLOWING GASTROENTEROSTOMY GASTRECTOMY

Dr. Constantine J. MacGuire presented a man forty-three years of age who was admitted to the Medical Division at St. Vincent's Hospital October 29, 1929. He had been operated on in the hospital two years previously for chronic duodenal ulcer. The gastroenterostomy performed at that time was followed by freedom of symptoms and progressive gain in weight and strength until nine months previous to this second admission, when he was suddenly seized with weakness, repeated vomiting of bright red and dark blood, tarry stools and also stools containing bright blood, and marked epigastric pain at about the umbilical level just to the left of the mid-line. After two weeks in bed he was relieved until one month before admission, when he collapsed with a recurrence of all the former symptoms.

An X-ray series showed evidence of obstruction and inflammation beyond the gastroenterostomy stoma with a six-hour retention. At operation, November 19, 1929, there were found at the first portion of the duodenum a cicatrix, causing almost complete constriction at the pylorus, and a very small duodenum. No evidence of recent or acute ulceration in the duodenum. About 2 centimetres beyond the gastroenterostomy stoma there was an ulcer which had perforated the wall of the jejunum and the posterior wall of the transverse colon, forming a jejuno-colic fistula about 1 centimetre in diameter, well protected by peritoneal adhesions. Surrounding this ulceration and fistula there was much induration in the adjoining wall of the colon and jejunum, extending to and involving the gastroenterostomy stoma. There were many enlarged inflammatory lymph-nodes and there was induration and chronic inflammation extending down to the mesentery of the jejunum, up into the mesocolon and the lesser peritoneal sac. The stomach was dilated. Since repair of the jejuno-colic fistula would require resection of the jejunum and the obliteration of the old gastroenterostomy stoma, subtotal gastrectomy seemed indicated. The entire involved portion of jejunum was then resected and the lumen restored by end-to-end anastomosis. A subtotal gastrectomy was then performed, end of stomach to side of jejunum, well beyond the resected area. More than the distal half of the stomach was resected. Because of the fact that the field had been contaminated by the operation on the fistula into the colon, the wound was drained.

A few days after operation a fecal fistula manifested itself, but under open treatment with dry heat this closed up in a couple of weeks, and since that time patient has been free from symptoms referable to his intestinal tract.

Check-up X-rays were taken about two weeks ago and were interpreted as follows: "An examination of the stomach to determine its emptying time after operation revealed the following: The remaining portion of the stomach

was dilated, its pyloric segment and a portion of the middle pole having been resected in the operation of November, 1929. The anastomosed area between the gastric stump and the jejunum was fairly well visualized and the barium mixture passed from the stomach into the dilated jejunum within the normal six-hour period. At the end of six hours the bulk of the barium mixture was in the proximal colon and the head of the column was entering the transverse colon. Small traces of barium could be seen in the upper abdomen, probably in the region of the proximal jejunum."

This case is shown as one of the unfortunate and apparently unavoidable results occasionally following gastroenterostomy, where the original lesion is cured only to be replaced by a very much more serious one. The mortality of jejuno-colic fistula is high. The diagnosis in this case was aided by the presence of fresh blood in the stools as well as in the vomitus; the great severity of the pain and its location just to the left of the umbilicus. The subtotal gastrectomy was done anterior to the colon because this operation does not constrict the colon, and in case of further trouble the anastomosis is easily accessible directly under the anterior abdominal wall.

JEJUNAL FISTULA TO ANTERIOR ABDOMINAL WALL

DOCTOR MACGUIRE presented a woman who was admitted to the First Surgical Division of Bellevue Hospital on January 14, 1931. She had been operated upon at the Presbyterian Hospital by Doctor Lambert in 1921 for a pre-pyloric ulcer. He performed a pylorectomy followed by an anterior Polya anastomosis, end of stomach to side of jejunum. A year later she returned to the Presbyterian Hospital suffering from symptoms which were judged to be due to jejunal obstruction, and for this Doctor Whipple performed a jejuno-jejunostomy between the two arms of the jejunal loop of the anastomosed portion of the jejunum. This was followed by relief of all symptoms until three years ago, when she developed recurrent attacks of pain relieved by food and sodium bicarbonate.

In the last year, the pains had been just above the umbilicus, and without definite relation to food, but relieved somewhat by local pressure. The pains had been getting worse, and for four months before the last admission she had failed to get relief. Operation was performed January 21, 1931. Very massive organized adhesions were found between the stomach, liver, jejunum, transverse colon and anterior abdominal wall, just beyond the gastrojejunal stoma, and proximal to the jejuno-jejunostomy of eight years before. There was a perforated jejunal ulcer adherent to the anterior abdominal wall, where there was a walled-off perforation, the base of the crater being in the anterior wall.

With much labor the ulcerated jejunum was freed from the anterior abdominal wall. The jejuno-jejunostomy of eight years ago was left intact, but the afferent and efferent loops of jejunum going to the anterior gastrojejunostomy of eleven years ago were divided between clamps, and the jejunal opening closed. The distal two-thirds of the stomach were then removed and the jejunum anastomosed to the open end of the stomach in front of the transverse colon in the usual manner.

This case illustrates the possibility of jejunal ulceration following a Polya operation when only the pylorus is removed. It should be noted that the jejunal ulcer develops subsequent to the jejuno-jejunostomy, an operation which has the obvious drawback of side-tracking the alkaline duodenal contents from the gastrojejunostomy stoma where it is so necessary for the neutralization of the acid secretion from the stomach.

The incidence of jejunal ulcer following stomach operations is certainly greater than was formerly thought. Secondary operations form a very real problem, and although total gastrectomy as a primary operation may be open to criticism, there is no question of its indication in these secondary cases.

Dr. Howard Lilienthal called the attention of the society to the frequent results of the removal of a large portion of the stomach with the idea of getting rid of the acid-bearing field. Recently in Philadelphia at a meeting of the American Surgical Association, it was shown that if any gastric mucosa at all is left, acid-bearing tissue will remain.

Dr. Richard Lewisohn said that the amount of stomach to be resected depends on the size of the stomach. This varies a great deal. The distal half of the stomach should be removed in order to prevent recurrences. In the first case presented, less than one-third of the stomach had been removed, according to the röntgenogram. Therefore, this patient may be subject to a recurrence at a later date. In the second case, pylorectomy had been performed as the primary operation. It is well known that a pylorectomy does not change the acid figures and thus will not safeguard the patient against secondary ulcers.

PAPILLARY ADENO-CARCINOMA OF THYROID—CHRONIC STRUMITIS

Dr. Charles Gordon Heyd presented a woman, forty-six years of age, who entered the Post-Graduate Hospital October 26, 1930, complaining of goitre, nervousness and loss of weight. She began to notice an enlargement on either side of the mid-line of the neck about four years ago. For three years there was no appreciable increase in size, but last year it had become noticeably larger. During the past three months the patient has become slightly hoarse. Basal metabolic determinations were 8 per cent. above the average normal. October 29, 1930, the patient was operated upon. The right lobe of the thyroid was approximately 6 by 4 by 4 centimetres, densely hard, encapsulated except for a small amount of normal tissue posteriorly. It had remarkable friability but was still definitely within the thyroid capsule, and while the friability and general appearance suggested malignancy, the absence of any perforation of the capsule seemed to render the diagnosis of strumitis possible. The left side was approximately 4 by 4 by 6 centimetres and represented a hypertrophy with generally normal characteristics. The operation was conducted through a typical thyroid exposure.

Section of the masses removed showed small lobules of thyroid gland separated by slightly thickened connective tissue. The lobules were made up of rather small thyroid alveoli, most of them containing well-formed colloid and lined by low cuboidal epithelium. In the stroma between the lobules there were very abundant lymphocytes occurring in numerous dense collections and in some places the bands of fibrous tissue attained a thickness of a millimetre. There had evidently been a definite inflammatory reaction in the stroma.

Microscopic sections of the nodule presented a stroma of dense fibrous tissue in which there were highly irregular groups of thyroid alveoli. Many of these were elongated, branched and partly filled by papillary epithelium. These atypical epithelial cells invaded the fibrous capsule at the margin of the nodule and extended practically to the

more normal thyroid tissue. There were moderately numerous mitotic division figures in the epithelial cells of these atypical thyroid alveoli. In some places there were abundant lymphocytes in the stroma about the alveoli. *Diagnosis.*—Papillary adeno-carcinoma.

The patient made an uneventful recovery and was discharged from the hospital on December 5, 1930, and in the interval since the discharge from the hospital has had a course in deep X-ray therapy.

Dr. Morris K. Smith said that it is disconcerting to remove what is thought to be an adenoma and to receive a pathologic report of carcinoma. He remembered such a case in which, four or five years after operation, no recurrence of the disease was found unless an enlargement of the spleen could be so construed. Another patient reported by the pathologist as having carcinoma developed what was thought to be a local recurrence. She was treated by X-ray and at present, five years and more after the operation, has no sign of malignancy, although she has to take thyroid extract.

Dr. Charles E. Farr said that he had had experience with five cases of carcinoma of the thyroid and had seen one other. His own five patients all died very promptly regardless of operation or radium therapy. Four were proved to be carcinomatous by operation or biopsy. One died without operation but was clinically surely malignant. The sixth case, not his, is still alive but has a recurrence.

Dr. William C. White said that last November he operated on a man for an enlarged thyroid gland and on going down found pale gray material, friable, not vascular, and easily removed. The pathologists disagreed as to the nature of the growth. It was diagnosed by different men as lympho-sarcoma, small-cell carcinoma, and Riedel's struma. All thought it was sensitive to irradiation. He was given high voltage Röntgen therapy without improvement. The man died in January with marked recurrence.

Dr. Alexis V. Moschcowitz said that for many years he had noted that there existed two varieties of carcinoma of the thyroid; one, in which the surgeon makes the diagnosis of carcinoma of the thyroid pre-operatively. These are the cases that do badly, a great many of the patients dying of metastases or local recurrence. In the second group, the surgeon removes the gland or an adenoma, which, upon examination, the pathologist reports as carcinoma. These cases usually do well, a majority of them having no further symptoms from the carcinoma.

Doctor Heyd, closing the discussion, expressed the opinion that the goitre patient he had presented would have recurrence. He was quite in accord with Doctor Moschcowitz that there was a great deal of clinical difference between the patient with a goitre who had a pre-operative diagnosis of malignancy and the patient with a goitre who had a histologic diagnosis of malignancy after operation. The former patients did badly whereas the latter did well. It was also well to bear in mind that there is another type

of malignancy which springs from the ultimo-bronchial body which is highly malignant and which at operation exhibits a growth behind the carotid artery. This anatomic relationship does not exist in malignancies that spring from adenoma.

ADENO-CARCINOMA OF THE RECTO-VAGINAL SEPTUM

Doctor Heyd presented a woman, fifty-five years of age, who entered the Woman's Hospital August 5, 1929, complaining of bleeding from the vagina of three weeks' duration. Examination revealed a recto-vaginal fistula 1 centimetre in diameter, with a cauliflower growth extending from the primary site in the rectum into the vagina. She had had bleeding from the rectum for the previous six months, supposed to be due to hemorrhoids. Three weeks previous to admission she noticed a thin, watery, odorless discharge from the vagina and then some bright red blood. A vaginal examination revealed a malignant process at the junction of upper and middle third of recto-vaginal septum with an indurated sloughing mass 4 centimetres in diameter, the mass sloughing both inside the rectum and inside the vagina. The Wassermann reaction was negative. Under gas anæsthesia a biopsy was performed with the histologic diagnosis of papillary adenocarcinoma. August 16, 1929, under anæsthesia, six needles, each containing 12 milligrams of radium, were inserted into the mass. The patient was discharged from the hospital on August 23 and readmitted February 3, 1930. Since her discharge from the hospital the patient had experienced increasing difficulty in moving her bowels. Rectal examination at this time showed considerable induration on both sides of the rectum. February 11, 1930, five needles, each containing approximately 12 milligrams of radium, were inserted into the posterior vaginal wall 1 centimetre apart, one in the midline and two laterally on either side, and a tube of 101.2 milligrams of radium was inserted into the rectum through a proctoscope so as to rest in the centre of the annular carcinoma. In addition, during this time the patient received 8 deep X-ray treatments. March 7, 1930, a permanent colostomy was performed by Dr. George Gray Ward, using the Sistrunk technic. The patient was discharged from the hospital April 25, 1930, with colostomy functioning perfectly and with some shrinking of the tumor.

She was readmitted June 23, 1930, and a modified Kraske operation with resection of the entire posterior wall of the vagina was performed by Doctor Heyd July 2, 1930. The gut was freely mobilized and resected *en masse* at about the junction of the recto-sigmoid and rectum, removing thereby the rectum proper and the anus and both sphincters intact. The lower end of the sigmoid was inverted by three rows of sutures and reinforced by interrupted sutures of linen. The peritoneal cavity was sponged dry and hermetically sealed with running suture of No. 2 chromic catgut. Following this a posterior vaginal wall was constructed by suturing the divided vaginal flaps. A self-retaining catheter was placed in the bladder and a small strip of iodoform gauze in the vagina. After the reconstruction of the vaginal wall the peritoneal body was reconstructed by the apposition of the levator ani beneath the posterior commissure, following which a considerable portion of the anatomic hiatus was obliterated by approximating the loose tissues and the wound closed with interrupted silkworm sutures. Anæsthesia consisted of .6 gram of sodium amytal, intravenously; narcosis produced in four minutes; plus spinal anæsthesia. Drainage consisted of the following: (1) Self-retaining catheter in the bladder; (2) iodoform strip in the vagina;

(3) cigarette drain in the area of the perineum; (4) two strips of rubber tissue and one iodoform gauze pack in the hollow of the sacrum.

The specimen from the Kraske operation consisted of the rectum, which measured 16 centimetres in length. The anus, perineum and lower part of the posterior vaginal wall appeared normal. At the upper end of the vaginal wall a fistulous opening 3.5 centimetres in diameter, with sloughing, rigid edges, led into the rectum. The specimen was opened after fixation. The rectum from the external to the internal sphincter seemed to be intact. The internal sphincter was considerably bulged toward the lumen, apparently due to carcinomatous infiltration. The ampulla recti was mostly occupied by a carcinomatous mass by projection from the lateral portions of the rectal tube, anteriorly leaving a narrow channel between the posterior wall of the rectum and a wide "V"-shaped space between the anterior rectal wall and posterior vaginal wall respectively. The area of the fistulous opening had a diameter of 2.5 centimetres posteriorly. The rectal wall seemed to be well preserved above and below the fistula.

Microscopic examination showed a typical adeno-carcinoma of primary alveolar, secondary solid type. It invaded in large plump clusters and extended into distant tissues by a diffuse dissemination of single cells or short cell rows. The region of the anus was likewise occupied by the carcinoma. Here it propagated by large atypical cells diffusely along the tissue spaces up to an area which was close to the normal squamous surface epithelium. The glandular structures were irregular, with multiple central necrosis. The epithelial rows were stratified, the cell outlines were indefinite. The stroma between the carcinomatosis structures showed only moderate inflammatory reaction. A large number of eosinophilic cells were found among the cells of inflammation. Final pathologic diagnosis was adeno-carcinoma of the rectum, extending into the vagina: rectovaginal fistula.

The patient had a very stormy convalescence. Owing to the short distal loop below the colostomy, there was leakage through the posterior sacral wound with considerable loss of substance from sloughing, which was unusually slow in granulating in.

For a considerable period of time the patient made no general systemic headway, requiring a series of transfusions to bring back tissue tone and recuperative power. She slowly gained in weight and was discharged from the hospital October 6, 1930. From this time she has uninterruptedly improved with a gain in weight and strength and is able to function at her housework. She has a normal acting colostomy but has still a small persistent sinus in the posterior median raphe.

MELANO-CARCINOMA OF RECTUM

Doctor Heyd presented a woman, fifty-four years of age, who entered the Woman's Hospital June 10, 1930, complaining of rectal bleeding for the previous three years. She has had no pain but slight rectal burning. She has passed bright and dark red blood at various times. At biopsy on June 11, 1930, a carcinomatous nodule the size of a walnut was found arising from the right side of the rectum, 5 centimetres from the anal margin. The specimen was removed with radio knife. Six radium needles, each containing approximately 12 milligrams of radium, were implanted into the base of the tumor for twenty hours.

The biopsy mass consisted of two portions, one was composed of a solid tumor mass which appeared to be a broad compact layer of tumor cells. The protoplasm is transparent and slightly eosinophilic. There are numerous mitotic figures. Scattered throughout the mass are pigmented cells of very large size and irregular shape, bloated with coarse, mostly round particles of pigment which do not give a positive iron reaction.

The pigment was dark brown, almost black. The destruction of the melanophores was densest at the base of the papillæ. In some areas there were destruction and necrosis. The chromatophores appeared in dense aggregations, being the only remainders of the papillary structure. The other portion of the mass consisted of the same type of tumor cells but the papillary arrangements could not be detected, the cells being in compact masses. Two small glandular structures of the intestinal type were found imbedded in the alien cells, within a short distance from each other. Close to this area was a third stretch of inflamed intestinal mucosa. Invasion of the blood-vessels could not be detected. The histologic diagnosis was carcinomatous melanoma of the rectum.

Following the biopsy and radiation the patient was discharged from the hospital June 19, and was readmitted October 8, 1930. A permanent colostomy after the Sistrunk technic was performed by Dr. George Gray Ward October 10, 1930. Four weeks later a modified Kraske operation with resection of the posterior vaginal wall was performed by Doctor Heyd, November 13, 1930. The growth extended in a linear direction from the anal margin approximately two and a half to three inches upward, but there were evidences of glandular deposits at this area.

A radical excision *en masse* was made. The entire rectum, anus, and posterior vaginal wall were removed *en masse*. There was some slight soiling of the field owing to the perforation of the perirectal tissues, as above indicated. With the tumor mass and upper rectum removed, the lower end of the sigmoid was closed. The stump of the bowel was left free in the operative hiatus. The peritoneal cavity closed intact and completely with No. 2 chromic catgut sutures. The posterior vaginal wall was reconstructed with interrupted sutures of No. 2 chromic catgut and a new perineal body created with three interrupted silkworm sutures. The lower portion of the levator ani was brought together beneath the posterior vaginal wall, the skin wound being closed completely with silkworm suture, except at a point opposite the normal position of the coccyx, which was used for drainage.

This patient made an uneventful recovery and was discharged from the hospital December 31, 1930. Since leaving the hospital she has gained in weight. Her colostomy functions adequately. She has enjoyed comparatively normal health since her operation.

Doctor Heyd, in presenting these two cases of resection of the rectum, called attention in the first case to the marked invasion of both the vagina and rectum, with a large rectovaginal fistula, and in the second case to the unusual type of malignancy:—a carcinomatous melanoma, or melano-carcinoma. The biopsy performed by Doctor Ward removed practically the entire melanotic tumor as was evidenced in the gross specimen presented. The section of the mass removed after operation showed a gradual extinction of the melanotic characters. Furthermore, it is to be noted that both of these cases were treated intensively with radium needles and by tube radiation together with deep X-ray therapy. In both cases the entire posterior vaginal wall was resected *en masse* with the tumor, necessitating the reconstruction of the posterior vaginal wall. This, technically, was not a matter of difficulty and the final result immediately after operation was not unlike that obtained after a perineorrhaphy. Both cases showed perirectal infection, which undoubtedly was a marked factor in the first case and to a less extent in the second case, in the prolonged wound convalescence.

Dr. Howard Lilienthal said that he understood that both these patients have a functioning colostomy. He referred to two women whom he presented before this society some time ago, one of whom had had an ordinary carcinoma of the rectum, and in the other a carcinoma involving part of the vagina. He had been able in operating on each case to draw down enough

bowel to implant it in the anal region so each patient, by twisting axially (Gersuny's procedure) has good control and they were saved the annoyance and discomfort of a colostomy. Doctor Lilienthal said he mentioned this at this time because so many things are forgotten as time goes on. Every now and then one sees a case of inoperable carcinoma of the rectum that gets well with radium therapy. This did not mean that radium should be substituted for surgery but it should be remembered in cases which were inoperable. He referred in particular to a case of a man with diabetes who came to him with carcinoma of the rectum which had invaded the posterior wall of the bladder. Doctor Lilienthal proposed establishing a colostomy but the patient objected and was sent to Dr. Douglas Quick, who took a specimen which was examined by Ewing, who confirmed the diagnosis. This man got entirely well under radium therapy. There was no metastasis and he remained well for six or seven years and died with a different disease. The end justifies the means. Doctor Lilienthal mentioned this as presenting one ray of hope on an otherwise hopeless situation.

CARCINOMA OF THE TRANSVERSE COLON—RESECTION OF TRANSVERSE COLON AND SMALL INTESTINE; SEVEN YEARS POST-OPERATIVE

Doctor Heyd presented a man, aged fifty-nine years, who entered the Post-Graduate Hospital February 3, 1924, complaining of indigestion. For the previous two years he had suffered from crampy pain in the right upper quadrant, coming on three to four hours after meals, and accompanied by nausea and vomiting. He was usually relieved of his pain by vomiting or by the belching of gas. His history was otherwise unimportant. There was tenderness slightly to the right of the median line. Gastro-intestinal X-ray examination showed an infiltrated lesion at the mid-point of the transverse colon. February 4, 1924, he was operated upon. At the median point of the transverse colon was an annular carcinoma which obstructed the lumen of the bowel to a degree of approximately 90 per cent. On the anterior surface there was a leakage or perforation with adhesions of the ileum and infiltration into the wall of the ileum. There was considerable inflammatory reaction in both the ileum and in the transverse colon. The small intestine in the region of the cæcum was bound to the anterior abdominal wall as the result of former appendectomy. The gall-bladder was chronically diseased and thickened, and had lost its color, but was without calculi. The stomach and duodenum were apparently clear. No abdominal metastases. No liver metastases. There might possibly have been metastases along the border of the pancreas but these were not palpable on account of the mesenteric fat. An atypical resection of the transverse colon and attached small intestine was done. Gastro-colonic omentum was divided, as was the mesocolon, and the tumor mass mobilized. This involved a resection of the mid-portion of the transverse colon about six and a half to seven inches in extent and a resection of nine inches of the small intestine where the growth had encroached and grown into the small intestine. The operation was technically difficult on account of the former adhesions and the loose, friable fat. The small intestinal ends were united in typical end-to-end anastomosis, two-layer technic. The same technic was employed for anastomosis of the transverse colon but had to be modified somewhat on account of the narrow

lumen of the distal portion of the transverse colon. The gall-bladder was left undisturbed. The adhesions were left undisturbed. Three strips of rubber tissue, one to the area of Morrison's space, and two perforating the omentum and carried down into the neighborhood of colonic anastomosis. The omentum was placed between the colon anastomosis and the small intestinal anastomosis.

The pathologic specimen comprised one loop of large intestine to which a loop of small intestine was adherent near the tænia. The large intestine was 110 millimetres long and the loop of small intestine 120 millimetres long. The lumen of the large intestine was markedly narrow, so far that only a forceps could be passed. The mucous membrane in the area of the narrowing was transformed into a fungoid mass and the entire wall of the intestine was infiltrated by a friable yellow mass. The lumen of the small intestine was wide open. The mucous membrane was regular but there was one nodule which projected into the lumen which was covered by mucous membrane.

Microscopic section showed one specimen of large intestine. The superficial areas were composed of irregular large glands which were lined by cylindrical epithelial cells which showed marked variability in size and shape and frequent mitotic figures. The stroma between the glands showed round-cell and leucocytic infiltration. These glandular structures diffusely infiltrated the entire wall of the intestine and were found near the serous surface. There were large areas of necrosis and the wall showed infiltration by lymphocytes, leucocytes and large wandering cells. A second piece showed mucous membrane of the small intestine with villi. The wall of the small intestine was infiltrated by the glandular structures as described above which penetrated through the entire structures and were found very near the epithelial surface. *Diagnosis.*—Adenocarcinoma of the large intestine infiltrating a loop of small intestine.

The patient was discharged from the hospital March 3, 1924, with wound entirely healed and general condition excellent. From the time of his discharge he has enjoyed uninterrupted good health; has gained some 30 pounds in weight. Three years after operation he had a complete X-ray examination of the gastro-intestinal tract which was reported negative. He has remained in perfect health from the time of his discharge from the hospital and in the succeeding seven years there has been nothing to suspect he has had any recurrence of his former trouble.

CHRONIC CYSTIC MASTITIS; ITS RELATIONSHIP TO CANCER

Dr. Otto Pickhardt read a paper with the above title.

Dr. Geo. L. Rohdenburg said that Doctor Pickhardt had demonstrated that in chronic cystic mastitis the histologic appearance does not always go hand in hand with the clinical course of the disease. The pathologist recognizes a normal architecture of the breast; in cancer he sees a disordered architecture. Under the microscope chronic cystic mastitis morphologically stands in the mid-position, and having seen the last stages as cancer, the histologist becomes suspicious of the architectural arrangement and calls it pre-cancerous. Unfortunately, the histologist cannot say what the biology of these cells is going to be. A similar condition exists with what are known as "carcinoids" of the appendix. It was a considerable time before it was recognized that these lesions were usually clinically benign. Experimentally, analogous degrees of proliferation have been produced by Schlarch "R," and these have been shown to be clinically benign. With coal tar, the histologic appearance is much the same, but the clinical course is vastly different.

Dr. Howard Lilienthal believed that sometime the day would arrive when the term "pre-cancerous" will no longer be used any more than the expression "threatened with typhoid fever" is now being used. The patient has cancer or does not have it. If in doubt, operate as if it were cancer; if there is no doubt and if the pathologist says there is no cancer there is nothing to worry about even if he says it is "pre-cancerous." Chronic inflammation or ulceration demands appropriate treatment without making a diagnosis of "pre-cancerous" lesion. There is, indeed, a great deal in the mental relief of these patients. The speaker was certain from experience that sometimes the removal of a specimen in an organ like the breast is unwise and it is better to proceed on the history and clinical findings, the idea being that a breast may show no cancer in the specimen removed and later on another part will show malignant disease. If in doubt, take off the breast.

Dr. DeWitt Stetten, on December, 1928, made the statement that in a relatively large material, mainly among private patients, whom he had been able to follow up rather carefully, he had never seen a case treated primarily for chronic cystic mastitis which had become carcinomatous and, since that time, nothing has occurred that would cause him to modify that statement. Doctor Stetten believes that chronic cystic mastitis is a benign condition, and, in spite of the general impression to the contrary, he does not think that there is any etiologic connection between chronic cystic mastitis and carcinoma of the breast. Practically all of his cases were treated by conservative measures—namely, excision of the cystic mass. In his whole series only two simple mastectomies were done, and these both on the same patient. He has never done a radical mastectomy for chronic cystic mastitis. In those cases in which the cyst alone was excised, the microscopic examination of the surrounding breast tissue almost invariably showed further evidence of chronic cystic mastitis. A number of the cases had recurrences of the disease, requiring in many instances two or more operations, but microscopic study never showed malignancy. Doctor Stetten is so convinced of the benignity of chronic cystic mastitis that, even if the pathologist's report is "pre-cancerous," he treats the case conservatively, if the gross appearance of the lesion is not suspicious. Long observations on a number of such cases over periods running up to ten years have as yet failed to show any development of malignancy. Doctor Stetten agrees with Doctor Lilienthal that the term "pre-cancerous" should be discarded from our nomenclature, as a lesion is either cancerous or it is not. While all admit that chronic irritation may be one of the factors in the development of carcinoma, apparently chronic cystic mastitis is not such an irritation. Of course, carcinoma may develop in a breast invaded with chronic cystic mastitis, just as it may develop in a normal breast, but there is no more evidence to prove that chronic cystic mastitis is a "pre-cancerous" lesion than there is to prove that a gastric ulcer predisposes to carcinoma of the stomach. It is quite true that in many cases of carcinoma of the breast, chronic cystic mastitis will also be found, but this is

readily accounted for by the fact that the ages at which the two diseases are prevalent are about the same. Although the lesion is a benign one, definite cysts or nodules should invariably be excised, certainly when they first occur, as a definite diagnosis cannot be made without excision and the patient will only obtain mental relief by the removal of the lesion. In some cases Doctor Stetten has ignored small recurrences if they are typical on physical examination and if the diagnosis has been definitely established by previous excision.

Dr. Alexis V. Moschcowitz stated that he agreed that the term "precancerous" does not mean anything. The patient either has a cancer or he has not. If the patient has no cancer, even a simple mastectomy is not indicated. Doctor Moschcowitz sees a great many patients upon whom he makes the diagnosis of a chronic cystic mastitis. It is his habit in these cases to reassure the patient but not to advise any particular treatment.

In doubtful cases, he advises a biopsy by means of a frozen section. If the report shows the presence of a chronic cystic mastitis, he abstains from any further procedure.

STATED MEETING HELD OCTOBER 28, 1931

The President, Dr. John Douglas, in the Chair

SUBMAXILLARY CALCULI

Dr. Russel H. Patterson presented five cases as follow:

Case I.—A woman, aged sixty, was first seen in Bellevue Hospital October 14, 1931, complaining of a lump in her throat near the mouth which had been present for four years. When she eats or chews she has a lump in her neck, which she has always been able to push back into the mouth and have it remain there, but during the last two weeks she has had constant pain with an increase in size of the swelling in the neck. She is a very large, obese woman. She presents a hard, painful swelling one and one-half inches in diameter in the right sublingual region which increases in size on chewing and swallowing. There is a pea-sized calculus felt at the entrance of the right sublingual duct which seems to completely obstruct the duct. *X-rays* show two calculi about one-half centimetre in diameter in the floor of the mouth in the region of the right sublingual duct.

Treatment.—With a mouse-tooth forceps a stone was removed from the lingual caruncle. The second stone required a small incision but was easily removed, as it was not more than one centimetre from the lingual caruncle. The anæsthetic consisted of applying 10 per cent. cocaine solution to the floor of the mouth. The patient is now rapidly recovering.

Case II.—A woman, aged thirty-eight, first seen April 12, 1930, complains of intermittent pain and swelling of the right side of the floor of the mouth over a period of several weeks. There is a definite swelling about the size of a hickory nut in the region of the right submaxillary gland. *X-rays* showed a shadow approximately three by six millimetres and with the long axis perpendicular. This shadow is about two centimetres anterior to the angle of the mandible.

On April 15, 1930, patient under general anæsthesia, an incision was made in the back of the floor of the mouth on the right side, and by dissection a stone about the size of an English pea was removed from the substances of the gland. There were several fine pieces of gravel in the immediate vicinity which were also removed. Following the operation the patient had a moderately extensive nonsuppurating inflamma-

tion about all the tissues on the right side of the mouth and neck. This slowly subsided over a period of several weeks and the patient has been symptomatically cured since.

CASE III.—A man, aged thirty-six, was first seen December 2, 1929, complaining of a lump in the floor of his mouth on the left side. Examination shows a swelling in the region of the left sublingual gland about the size of an English walnut. The duct cannot be probed. There is a thickened ridge along the course of the duct, and about one and one-half inches from the entrance to the duct there is a hard pea-sized mass felt which is thought to be a stone in the duct.

X-rays show a rounded dense area about the size of a small French pea some one and one-half inches posterior to the canine tooth opposite the ramus of the left side of the jaw.

December 4, 1929, under cocaine, an incision was made parallel to the submaxillary duct down to the stone and the stone was enucleated. Convalescence was uneventful and he has had no further symptoms from the condition.

CASE IV.—A man, aged thirty, was first seen September 10, 1928, complaining of pain and tenderness in the right side of the floor of the mouth. The right submaxillary duct was bougied and a stone, one-half centimetre in size, followed the bougie out into the mouth. The patient was free of symptoms for one year when he again had pain and tenderness and swelling in the right side of the floor of his mouth. He has had intermittent pain, swelling and soreness of the mouth since.

X-rays taken recently show four calculi in linear antero-posterior arrangement, suggesting calculi either in the right sublingual or submaxillary salivary glands or in their ducts. Thus far none of them have passed. It is evident that an operation will have to be done to cure the patient.

CASE V.—A woman, aged fifty, was first seen on October 13, 1931, complaining of pain and swelling in the left side of the floor of the mouth. There is tenderness and a swollen area about the size of a hazel nut in the left side of the floor of the mouth in the region of the left sublingual gland. X-rays show a probable small calculus in this region. The left sublingual duct has had bougies passed in it on two occasions and a click has been felt, but thus far no stone has passed. The swelling in the gland has somewhat subsided. Operation will probably have to be resorted to.

The reporter said that exactly how these stones are formed is not known. They may be formed on foreign bodies, they may have as a nucleus bacteria or waste products from about the mouth. They are thought in some cases to form as a result of mercurialization. They are thought by some to form in patients with an arthritic diathesis or they may be formed by a change in the reaction of the salivary-gland secretion causing a precipitation of salts. They may vary in number and size. Hulke reported a stone weighing sixty-seven grams. They are more common in the ducts than in the glands and are more common in the submaxillary and lingual ducts than in the parotid duct.

X-rays are frequently reported negative for stone but such would not be the case if careful films were taken in the mouth, as a dentist would take a film of a tooth. The inflammation caused by duct obstruction must be differentiated from other infections, tumors and such conditions of the mouth and neck.

Treatment consists of: local applications, the passing of bougies in the ducts, and finally surgical intervention.

DR. CONDICT W. CUTLER, JR., remarked upon the frequent incidence of severe cellulitis in the submaxillary region, which would seem to indicate the

desirability of the early removal of these calculi as possible causes. As illustrating this fact he cited the case of a man who presented the picture of acute angina with a board-like swelling of the floor of the mouth. At operation a large stone, three centimetres in diameter, was discovered in the submaxillary gland. This was removed and the patient made a satisfactory recovery, but one year later he presented his doctor with a perfect cast of the submaxillary gland which he had himself removed from his mouth. He subsequently had no difficulty.

Dr. John Douglas related the case of a man who came to see him with a history of swollen glands in the right side of the neck for over three months which did not subside. Finally an abscess developed which was opened and drained, discharging a moderate amount of pus. But even after that the swelling continued in the floor of the mouth, making it difficult to swallow. The floor of the mouth became œdematous and swallowing became more difficult. A short time after this the patient felt a foreign body that looked like a piece of broom straw completely calcified which was sticking out of the floor of his mouth and which he pulled out. He then remembered that three or four months previously while eating cereal he had felt something stick in the floor of his mouth. Evidently that little piece of wood ran into the salivary duct. After it was removed the swelling completely disappeared.

Doctor Patterson reported that there was a woman in the wards of Bellevue Hospital who had a temperature of 104° as a result of extensive cellulitis of the floor of the mouth. Röntgenograms were negative, but physical examination revealed a stone emerging from the opening of the duct. Upon the removal of this stone simply by forceps pus gushed out and the patient's condition improved very much. Later the patient began to complain again. This time the X-rays revealed a second stone, which was removed. Then all symptoms subsided. These cases must be more common than is generally known for there are only about 400 which have been reported in the literature. At the time of Dr. Seward Erdman's paper in 1920 there were about 300 reported cases.

BILATERAL CHRONIC PAROTITIS

Dr. Russel H. Patterson presented a woman, thirty-four years of age, who was first seen September 15, 1931, complaining of a swelling in the front of the ears for two years. The onset of swelling was more or less insidious. She attributed it to catching cold while swimming. The swelling becomes alternately larger and smaller. First one side is painful and then the other. The left one was very painful a week ago but after a discharge of white-gray material the pain disappeared and the swelling became smaller. Now the right one is swollen and painful.

There is a swelling in the region of both parotids. The left parotid is soft and about twice its normal size. The duct is open and a clear serous fluid can be expressed. The ducts barely admitted a No. 1 Eustachian tube bougie. The right parotid is swollen and tense, about four times normal in size, and only the smallest amount of clear serous fluid can be expressed. No calculi felt. No inflammation of ducts can be demonstrated. She wears a partial

plate for upper and lower teeth. The tonsils are adherent and cryptic. In the neck some of the lymph-nodes at the angle of jaw are slightly enlarged. X-rays were negative.

Treatment.—Heat, massage and astringent mouth wash were ordered. The ducts have been dilated weekly until they now admit a No. 2 Eustachian tube bougie. The patient was told to massage the parotid regions gently every night. She also was advised to sip small amounts of weak lemon juice twice a day.

Other suggestions not yet carried out were: Use of diathermy; autogenous vaccine; use of X-ray therapy. Her condition has somewhat improved.

The case is presented because:

(1) Such cases are not frequently seen.

(2) Though the patient attributes the cause of the parotitis to a cold remarks from the members of this society as to whether such is likely or not are desired.

(3) Would bilateral partial stenosis be the cause, or an aggravating factor in the disease?

Dr. Willy Meyer suggested treating this condition by means of artificial hyperæmia with the help of an elastic neck band. This has resulted in much benefit in acute and in chronic inflammation of the parotid gland. It should be worn for many days, if necessary, ten to eleven hours of the day as well as night. There is hardly another inflamed tissue in the human body that responds as well to hyperæmic treatment as that of this particular gland.

Dr. William F. Cunningham said that eight years ago he operated on a woman who had previously been operated on five times for recurrent enlargement of the left parotid gland and discovered and removed a bristle from a tooth brush. The patient has had no further trouble. Another patient, a boy, in Bellevue Hospital, for three years had had chronic enlargement of the right parotid gland which had been incised three times. The diagnosis was thought to be tuberculosis of the preauricular lymph-nodes or actinomycosis. Sections taken were negative for actinomycosis and tuberculosis. The lower two-thirds of the gland was removed and on section multiple cysts were found.

On microscopical examination these cysts were found to be lined by stratified columnar epithelium and the walls contained lymphoid tissue. These factors are indicative that the disturbance was of branchial origin.

REPAIR OF DEFECT IN COLON BY TRACTION

Dr. Eugene H. Pool presented a man, thirty years of age, who was admitted to the hospital on December 22, 1926, with supposed thrombosis of the mesenteric vein. Onset occurred three days before admission with severe pain in the left flank and vomiting.

He was acutely ill, the abdomen distended. Patient was explored under local anæsthesia through a lower left rectus incision. An enormously distended, gangrenous loop of large bowel filled with gas and old blood presented. About three feet of this bowel were removed. The distal limb of the gangrenous bowel reached down into the pelvis, the proximal was gangrenous almost to attachment at descending colon. Tapes were loosely tied around the limbs

of the projecting intestine flush with skin; rubber dam inserted about each. Redundant bowel was cut away. Patient had also some turbid fluid in lower abdomen. Post-operative course stormy for first week. Gas and fæces began to escape from the proximal loop on the third day. The distal limb sloughed down to the recto-sigmoid junction and had to be trimmed out from day to day. The upper sloughed apparently to the descending colon. Barium enema showed about four inches of rectum remaining. An attempt was made gradually to approximate the proximal to the distal segment of bowel by traction. (Figs. 1 and 2.)

With this in mind while the wound was wide open and granulation tissue not firm a rectal tube was passed by anus into wound and sutured to upper segment which was loosened by blunt dissection as much as possible without opening into peritoneal cavity. The tube was attached to the thigh by a rubber band producing traction.

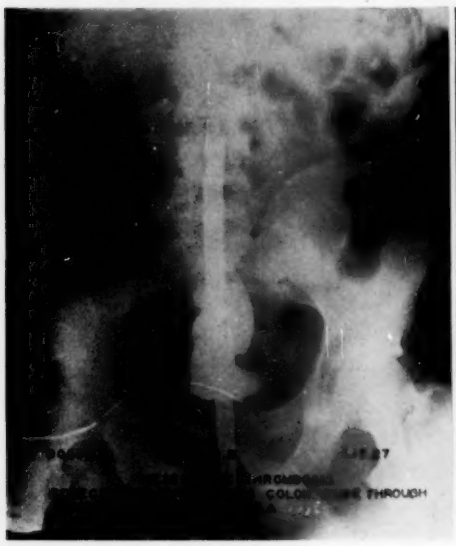

FIG. 1.

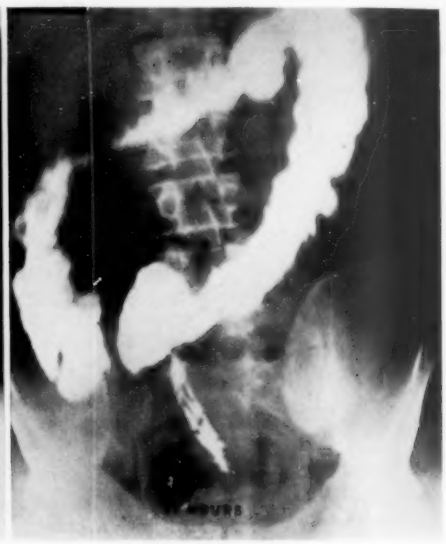

FIG. 2.

FIG. 1.—Twenty-five days post-operative.

FIG. 2.—Rectum and descending colon in contact. Rectum evacuated. Retention above showing stricture between the two loops. This was about two months after the original operation.

Ultimately by repeatedly doing this the ends were brought close together, bridging the gap which would have been about four inches filled with scar tissue and only to be corrected by some complicated plastic. Whether the bowel elongated or was displaced downward by the traction is, of course, a question. The procedure was carried out six times, sutures holding up to two days. He was discharged with a fecal fistula from a tear in the anterior wall of the bowel. Patient had been passing fæces per rectum for some days before discharge. May 6, 1927, a right-angled intestinal clamp was applied to an intestinal spur which was obstructing the lumen of the bowel. Following this fæces moved more freely by rectum. October 7, 1927, colostomy wound was closed and a temporary cecostomy done. The closure healed firmly and his fæces passed by rectum. Barium enema showed the constriction to be narrow but the barium passed through readily. The cecostomy closed automatically. April 4, 1929, returned because of acute intestinal obstruction. Under local anæsthesia exploration was made and tube inserted

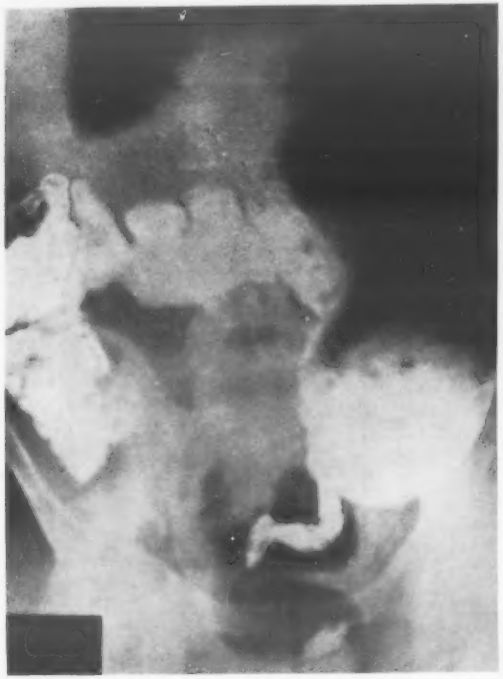

FIG. 3.—Three years later. Dilated upper loop.

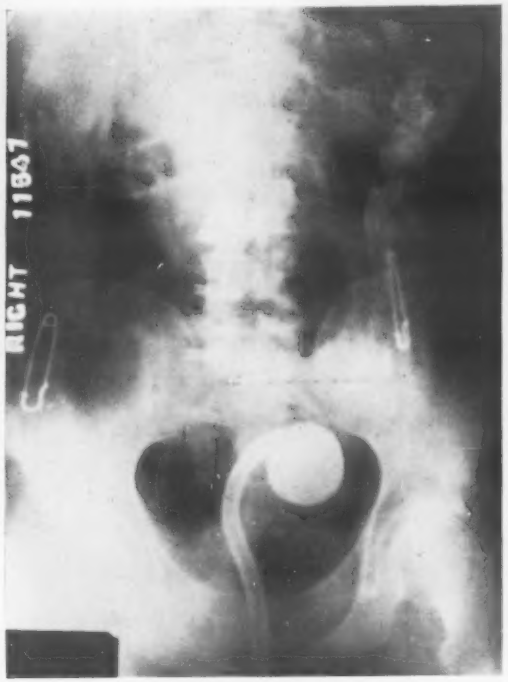

FIG. 4.—Murphy button in place. A sound in rectum.

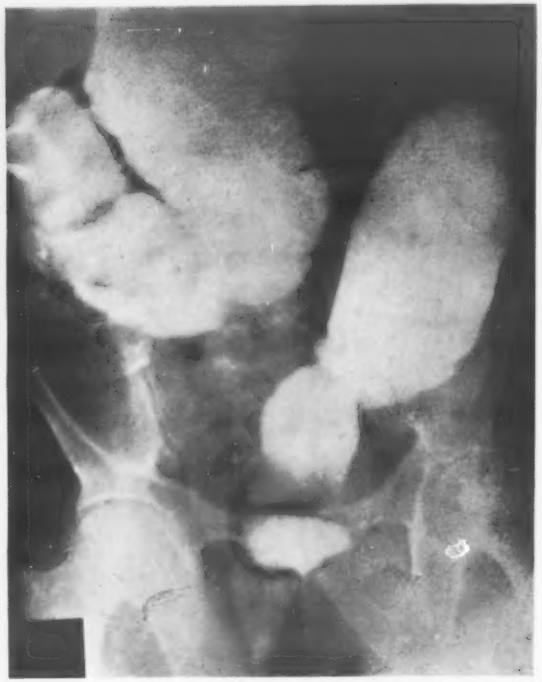

FIG. 5.—Final result. No stricture.

into large intestine just above site of obstruction. Convalescence smooth thereafter. Fistula closed spontaneously. November 19, 1930, diagnosis of incisional hernia and stricture of bowel. X-ray showed stricture at point of anastomosis between colon and rectum. Efforts to dilate with bougies were not satisfactory. The proximal loop was enormously dilated and overlapped the rectum. Laparotomy, stricture identified and Murphy button anastomosis done, adhesions freed and ventral hernia repaired. Post-operative course smooth, distension disappeared. Abdominal wound remained firm. He has had no further trouble. (Figs. 3, 4 and 5.)

This case was presented before the society because it calls attention to a principle which may be of value but has been generally neglected; namely, the potentiality of tissues to elongate or expand under continuous tension. This is seen in the enormous sacs of certain scrotal herniæ, and in the elongation and extension of the sigmoid in recurrent volvulus. Numerous other illustrations might be given. It has long been in the mind of the author that

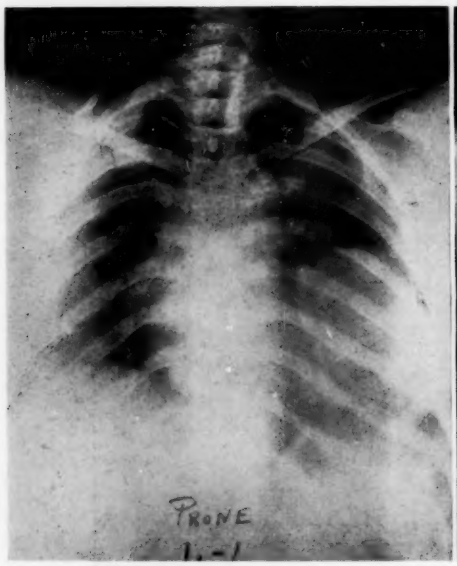

FIG. 6.—Serous cyst of thorax. Prone.

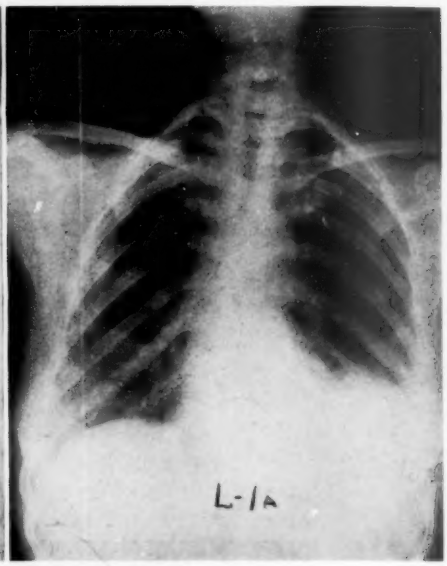

FIG. 7.—Serous cyst of thorax. Erect.

this property of tissues seen in such reaction to pathological conditions might be turned to advantage and deliberately utilized. The effort was made successfully in this case.

SEROUS CYST OF THORAX

DR. EUGENE H. POOL presented a woman of 56 years who was admitted April, 1931, because of pain in the epigastrium and left flank for four months. The symptoms became marked about two weeks ago with gradual onset of feeling of fullness and heaviness in epigastrium, at times sufficient to affect sleep. The lungs were clear except for a diminished resonance at left base with slightly diminished voice and breath sounds. The heart was not displaced and was normal. Fluoroscopical examination of chest showed on the left immediately above the diaphragm a sharply circumscribed area of increased density of a homogeneous character suggestive of lung tumor.

X-ray of chest showed this mass to vary in position with change in position of the body. In the upright position its upper limit lay at level of fourth

rib. (Fig. 7.) In the prone position at the level of the second rib. It lay in the mesial half of the left thorax in the upright position in front of the heart. In the prone position posteriorly. (Fig. 6.) It did not displace the heart. There seemed to be an irregularity of the diaphragm as if there were a connection between the thoracic and abdominal cavities. Bronchoscopical examination showed a small amount of purulent secretion in the bronchial tree. No sign of an intrapulmonary lesion. Lipiodol injection showed the mass to be extrapulmonary.

Patient was operated upon May 11. Intercostal incision fourth space with division later of fifth costal cartilage. Cyst was aspirated and contained 500 cubic centimetres of clear watery fluid. Its walls resembled pleura and the cyst lay in the angle between the pericardium and the upper surface of the diaphragm. It was attached to both the pericardium and the diaphragm through an area about three inches in diameter. Cyst incised and interior explored. The main part of cyst wall was entirely excised, but over attached area the

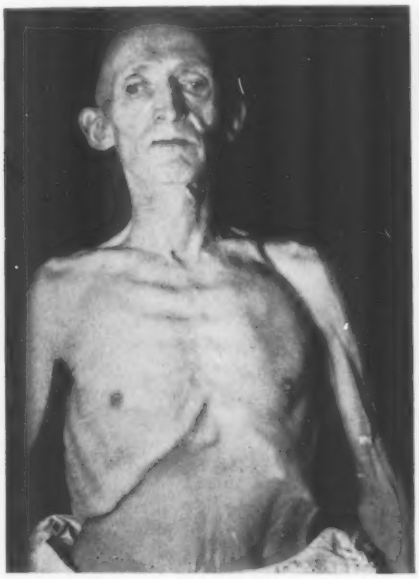

FIG. 8.—Showing emaciation on admission.

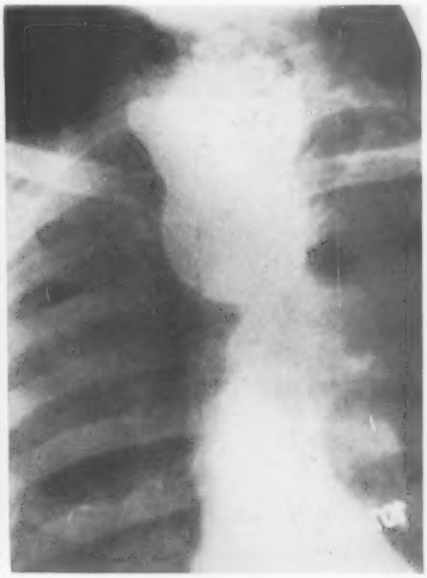

FIG. 9.—Diverticulum of œsophagus.

inner layer only was removed. This was readily done by blunt dissection. Wall entirely removed by the blunt dissection. Section of the cyst wall showed microscopically a loose fibrous structure with apparent covering cells of two to three layers of cuboidal type but nothing specific about the structure. The fluid contained a trace of chlorides and a heavy trace of albumin and slightly alkaline to litmus. Guaiac test was negative. The sediment from a centrifuged specimen contained a few large flat partially degenerated cells with nuclei. There were no crystals. Since the operation the patient has done well.

The case is presented on account of the rarity of such a lesion.

DIVERTICULUM OF ŒSOPHAGUS: TWO-STAGE OPERATION

DR. EUGENE H. POOL presented a man aged sixty-two years, admitted December 2, 1930, for vomiting, which first appeared five years before, and inanition. (Fig. 8.) He had been unable to retain anything during three weeks before admission. His best weight was 235 pounds ten years ago; one

year ago weighed 185 pounds; three days before admission weighed 134 pounds.

He was a chronically ill looking man showing great loss of weight. There was a bulging in the anterior neck. Diagnosis diverticulum of œsophagus. (Fig. 9.) Because of the malnutrition a gastrostomy was done on December 10, 1930.

Patient gained satisfactorily. Five weeks after the gastrostomy operation for diverticulum performed.

Incision parallel with anterior margin of left sternomastoid muscle. The thyroid gland on the left was exposed and rolled anteriorly and to the right. Stomach tube was passed until it met an obstruction. It was then felt lying in the diverticulum. The diverticulum was then drawn upward and delivered into the incision. A diverticulum of the œsophagus arising about the level of the cricoid process and the postero-lateral surface of the œsophagus and ex-

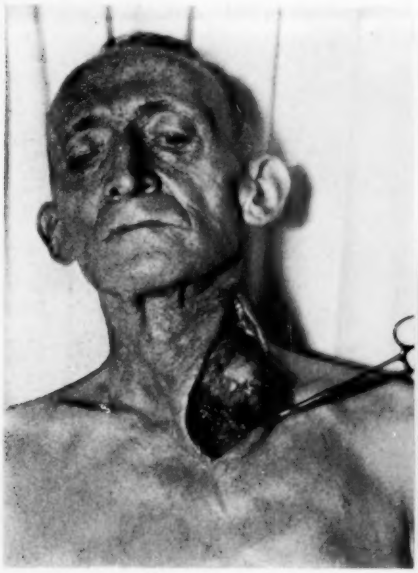

FIG. 10.—Second stage.

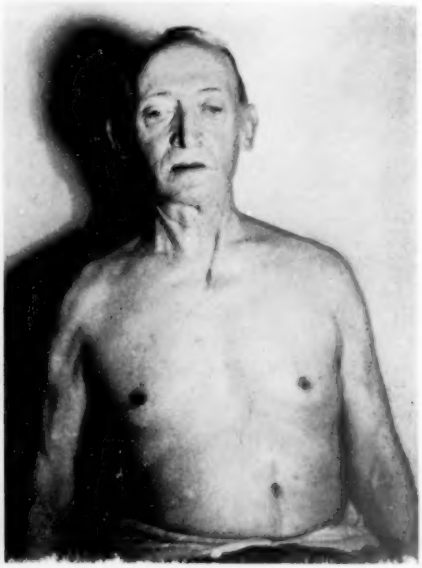

FIG. 11.—Condition three months after operation.

tending down into the thorax was found, measured about fifteen by eight centimetres. Walls were thick. The stomach tube was left in this sac and the adherent structures stripped free down to the neck of the sac. (Fig. 10.)

Excision of the diverticulum was done twelve days after this by Lahey's method. The diverticulum was elevated from its bed, its fundus amputated and the mucosa dissected free from the thickened muscularis to 1 centimetre from œsophagus. Packing was inserted into the stump of the amputated diverticulum. Gastrotomy tube withdrawn fifteen days after amputation of diverticulum and sixty-four days after its first insertion. Discharged seventy days after gastrotomy. Both wounds healed rapidly. Weight on admission 134 pounds. Weight April 17, 1931, 183 pounds. (Fig. 11.)

Subsequent note: November 14, 1931, No obstruction to bougie: X-ray shows existence of a small diverticulum which empties readily and causes no symptoms. This suggests the advisability of removing the outer wall more completely. This might be done at second stage by tying a heavy silk ligature around the whole sac about one centimetre from œsophagus after freeing and pushing inward the mucous membrane lining of this portion. The sac

would slough off beyond the ligature, the end of which would, of course, be left long.

Dr. Allen O. Whipple said that in cases where the diverticulum is small the procedure of delivering it well outside the neck is difficult. He operated recently on a patient with a small narrow diverticulum, but when the diverticulum was drawn up the top of it could barely be raised above the skin surface. It was anchored to the top of the incision without being opened and the patient was immediately relieved of the difficulty in swallowing. The granulated surface healed over and there has been no trouble since. In some of these cases, once the diverticulum is reversed and anchored in the vertical direction of the food passage, the second-stage operation may not be necessary.

Dr. Franz Torek said there was a difference of opinion as to the advisability of the one- or the two-stage operation in diverticulum of the œsophagus. Since Charles Mayo advocated the two-stage operation the majority of surgeons have been in favor of it. But on the other hand there are other clinics, such as the Jackson Clinic in Philadelphia, where the one-stage operation is done exclusively and in the speaker's own practice he has always done the one-stage operation. In favor of the two-stage operation the safety against mediastinitis is brought forward, and it is true that in the one-stage method greater care is necessary to avoid infection of the mediastinum at the time of operation and to prevent its occurrence subsequently. The former is accomplished by proper tamponing of the surrounding tissues, the latter by most accurate suturing, with the employment of very fine needles, such as are used in eye surgery.

In the one-stage operation there is the advantage of being able to determine with greater accuracy where the pouch should be cut off to restore the œsophagus to its original shape. Doctor Torek's method is very simple. Two fine guy sutures are introduced, one just above the neck of the pouch, the other below it. As these are held up, an imaginary line drawn from the one to the other indicates the site for the second line of sutures. The pouch is therefore cut off somewhat distal to this line, just far enough to permit the first line of sutures to be inverted. In the two-stage operation it is scarcely possible to estimate the correct site for ablation of the pouch with equal accuracy, unless one again exposes the œsophagus in its entire diameter, the very thing the advocates of the two-stage operation want to avoid. Consequently it may happen that too much is removed and that a constriction will result because the ablation was performed within the limits of the normal œsophagus, or else the difficulty of making an accurate suture in the granulation and scar tissue which has formed since the first stage may cause constriction due to excessive scar contraction at the suture line.

When Doctor Lahey read his paper on the treatment of diverticulum of the œsophagus, in which he advocated the two-stage method, he laid special stress on the necessity of regular bouginage after this operation, which in one case he continued well into the second year. This seems to point to the likelihood of stricture occurring after the two-stage method. In Doctor

Torek's experience with the one-stage method bouginage has not been necessary. He passes the bougie once only, after healing is complete, to assure himself and the patient that the passage is perfect, but never afterward.

Judging from Doctor Pool's röntgenogram bouginage will not be necessary in his case, as there is no narrowing. Here the opposite imperfection has resulted. For a reconstruction of the œsophagus too little has been removed, as the picture still shows the presence of a fair-sized diverticulum. If this happens in the hands of a master surgeon like Doctor Pool, it surely is not due to a lack of surgical skill; the fault should rather be sought in the method.

Lahey reports no mortality in his two-stage operation. On the other hand, in the series of one-stage cases reported by Jackson and Babcock there was likewise no mortality nor was there any in that of the speaker. So it seemed to Doctor Torek that the difficulty of guarding against infection by the two-stage operation is not so serious as reported, and he expressed the belief that the œsophagus can be more perfectly restored by the one-stage than by the two-stage operation.

Dr. John Douglas said that he had an experience similar to Doctor Whipple's. His patient was an old gentleman who could swallow nothing. An unsuccessful effort was made to introduce a Levine tube and it was suggested doing a gastrostomy to pass the tube from below. It seemed to Doctor Douglas that it would be easier to operate on the diverticulum under local anæsthesia and pull it up as a first stage. As soon as it was pulled up to a position where it did not point downward the patient could swallow and nothing further was done. He lived for several months swallowing easily, but eventually dying of a tuberculous condition. In a poor risk it would seem advisable to confine oneself to this without performing a second-stage operation.

Doctor Pool, in closing the discussion, stated that although Doctor Torek has had astonishing success in doing a number of one-stage operations without cellulitis, it cannot be denied that most surgeons view the one-stage procedure with deep misgivings because they have seen resultant severe cellulitis. The discussion this evening emphasized the question of treating a lesion rather than the patient. There should not be a fixed rule how to treat the œsophagus or any other part of the body; the individual indications should be weighed. This man, sixty-two years of age and starving, could not have stood much surgery nor infection; the fact that he has put on sixty pounds in weight, has returned to work, and is in perfect health is an indication of the success of the two-stage procedure. Whether he has had a perfect mechanical job is immaterial. Whether there is a pouch is likewise immaterial provided the pouch does not cause discomfort. The surgeon's aim should be to get the individual well, and this patient is well.

CHORDOMA. RADIUM TREATMENT—TEN-YEAR RESULT

Dr. Eugene H. Pool presented a woman whom he had presented twice before. (cf. Annals, vol. LXXVI, p. 123, 1922; and Annals, vol. LXXX, p. 157, 1924). Attention was called to the fact that the tumor is composed of syncytial cells (numerous nuclei but fused cell bodies without cell membranes)

and that it gave the impression of poor vitality which might well be susceptible to irridation. All reported cases at that time had been fatal, but rarely if ever did they show metastases, the development of the growth being by peripheral extension and infiltration. As nothing has been reported in regard to the late results of irridation, this case is of interest. It is now over ten years since the treatment which consisted in removing considerable of the sacrum and incomplete removal of the growth which produced within the sacrum a mass about four inches in diameter. Following operation she was treated at intervals for one year with radium at General Memorial. There is no sign of recurrence. The woman is seventy-five years old and decrepit from other causes.

CONGENITAL CYSTS AND FISTULÆ OF THE NECK

Dr. Herbert Willy Meyer read a paper with the above title for which see page 1.

Dr. Charles E. Farr said that he had had a moderate experience with these cysts of the neck of the congenital type. He once saw a case of bilateral sinuses of the neck with the lower openings just above the clavicles—one so inconspicuous as hardly to be seen except that an occasional drop of fluid appeared.

A week ago he operated upon a large cyst in the right side of the neck where by aspiration and injection of lipiodol an excellent outline of the cyst was obtained. A branch was seen running up to the base of the tonsil and another down the neck to within two inches of the clavicle. On examination of the photograph of this child a dimple could be plainly seen showing where the external opening at one time must have appeared. At operation the cyst was excised without difficulty. It was beneath the sternocleido muscle lying on the great vessels and the upper portion extending to the base of the tonsil. The lower ended blindly just short of the skin and two inches beneath the clavicle. Recovery was uneventful.

Doctor Farr said it had been his custom for many years in treating midline or thyroglossal cyst to remove a large portion of the hyoid bone, leaving a shell if possible. He then cored out the tract widely and deeply to the foramen cæcum. So far as he was able to follow up the cases all of them had remained healed to the present.

Doctor Meyer rejoined that he only wanted to add one fact. Branchiogenetic cysts as well as branchio-genetic fistulæ, of course, do occur, but they must lie above the level of the hyoid bone. Anything that lies below the level of the hyoid bone in the opinion of Wenglowski must come from the thymopharyngeal duct.

The idea of resecting the mid-portion of the hyoid bone was not original with the speaker, but he had felt the only way to cure these cases was to resect a portion of the hyoid and dissect the strand of tissue through to the foramen cæcum. The patient must be cured at the time of the first operation or a new cyst may develop if only a few epithelial cells remain. One must do a radical operation in order to surely cure these cases.

BRIEF COMMUNICATION

RUPTURED GANGRENOUS CÆCUM

EXTENSIVE inflammation of the wall of the cæcum is rarely met with. One finds references to diverticulitis of this portion of the bowel, but cases of phlegmonous involvement of the wall of the cæcum with abscess formation and rupture are very rare indeed. Search of the literature reveals but few such reports. (Bowen,[1] Hagler,[5] Hallopeau and Monod.[6]) For this reason, therefore, the following case history is deemed of sufficient interest to place on record by publication.

A white, female child, aged eight years, of good general health, during the night of March 2, 1930, developed pain around the region of the navel, unaccompanied by nausea or vomiting. This pain soon localized itself to the right lower quadrant. It persisted but did not increase when the patient had a bowel movement.

Seven days after the onset of the illness, the child still complained of pain in the abdomen; was not nauseated nor did she vomit; she was playful in bed; temperature 100.5°, pulse 94. Because of the relative playfulness of the child, because the temperature did not seem to rise more than it had been, and because the symptoms on examination did not become aggravated, it seemed that the acute inflammatory process was subsiding. Patient was still kept in bed on a light diet with icebag applications to the abdomen. Operation was constantly advised but refused.

On the tenth day, and for the first time since the onset of the illness, the child complained of pain on movement of the bowels, was nauseated and vomited. Temperature rose to 102.5°, pulse to 108. Catheterized specimen of the urine showed a faint trace of albumin; 15 pus cells to the field. Blood count 17,400, polymorphonuclears 86 per cent. When seen that evening, there was definite spasm and rigidity of both lower recti muscles, more especially marked over the right; the rebound tenderness was exquisite, the skin was hyperæsthetic over the right lower quadrant, Rovsing's sign was positive. There was no evidence of any mass. Again operation was strongly advised to which the parents finally consented. The operation was performed at the Royal Hospital on March 15, 1930, under open-drop anæsthesia. On admission the temperature was 101.6°, pulse 120, respiration 30. A definite mass was visible and palpable in the lower right quadrant of the abdomen, extending from the level of the umbilicus down to the pubis. A median right rectus incision was made. When the peritoneum was opened this mass presented itself. In attempting to free the portions of the intestines forming this mass, a localized abscess cavity was broken into and *B. coli*-smelling pus evacuated. This mass was composed of the transverse colon, a portion of the anterior inferior aspect of the colon being agglutinated to the medial aspect of the cæcum. When separated, it was found that there was a perforation in the medial aspect of the cæcum of 3 fingers' length and about 1½ fingers' breadth within the gangrenous cæcal wall. This opening in the cæcum extended nearly the length of the entire cæcum to just within a half inch of the ileocæcal junction. The anterior and posterior walls of the cæcum were infiltrated so that they were about a quarter of an inch thick, and the whole of the anterior and posterior aspects of the cæcum were gangrenous and the tissue very friable. The appendix was retrocæcal, acutely inflamed, apparently only by contiguity. That portion of the transverse colon which was adherent to the cæcum showed a contiguous gangrenous inflammation eroding the serous and muscular layers down to,

but not extending through, the internal mucosa for a distance of approximately 3½ fingers' breadth. Because of the poor condition of the patient, the pulse rate having risen to 148, respirations 60, and because of the extreme friability of the cæcal wall, it seemed best not to attempt any radical procedure. The rent in the external layers of the transverse colon was sutured over with interrupted sutures of catgut. The appendix was then removed. Because of the friability of the cæcal wall it was impossible to close the opening and one wide rubber tube drain was inserted into the cæcal opening, one tube to the pelvis and one to the lateral gutter toward the liver. The patient's immediate post-operative condition was one of severe shock.

For forty-eight hours following the operation, a proctoclysis of 5 per cent. glucose and 5 per cent. bicarbonate of soda was given by Murphy drip. The patient's pulse gradually dropped to 112, its volume and quality gradually improving. Temperature gradually dropped, so that on the third day it was down to 101.2°. The patient passed gas through the rectal tube. The dressings were saturated with purulent and fæcal material. She moved her bowels by rectum either with the aid of small enemata, or naturally. The drainage tubes were removed on the seventh day post-operative. The fæcal discharge continued for nineteen days after the operation. The wound and fæcal fistula were gradually closed over with granulation tissue. Healing was stimulated by the use of alpine light radiation. Seven weeks after the operation the wound was firmly healed; there was no evidence of any herniation.

Re-examination one year later revealed the wound to be still solidly closed, and the patient free of any complaints.

Comment.—Textbooks on pathology, embryology and physiology have little to say in detail of the adenoid tissue of the intestinal tract and especially the colon. Our works on diseases of the intestine are likewise careless in scientific details. Monographs on appendicitis speak freely of the adenoid tissue of the appendix, yet have no remarks on its analogue, the cæcum. There are abundant evidences that there are infections of the large intestine which have not been very clearly understood. Delafield and Prudden,[2] in their textbooks, refer to a very fatal and obscure form of necrotic colitis which appears to be septic in character. After death the inner surface of the colon is found studded with little blackish areas in which the blood-vessels are gorged with blood. The granular and connective-tissue coats are infiltrated with pus cells and there is a superficial necrosis. Various forms of microörganisms have been found in connection with suppurative and necrotic lesions of the ileum and colon—*streptococcus pyogenes, staphylococcus pyogenes aureus, bacillus coli communis, bacillus proteus, etc.* Ziegler[8] refers to inflammation of the large intestine as sometimes due to septic infection. Piersol[7] writes that the submucous layer of the cæcum, like that of the appendix, is rich in lymphoid tissue, which is readily subject to infection. Dowd[4] wrote that we have long known about inflammation of the large intestine which is known as colitis, which begins primarily in the mucous membrane, leading to a destruction of the parts of the membrane, and which sometimes involves the underlying coats. Through the lymphatics we get infection of this adenoid tissue (or follicular glands) of the cæcum. Irritating intestinal juices may produce erosions and with infection prevailing, these erosions may become ulcerated. If the blood stream is microbic or septic

and the cæcum sensitized, then hæmorrhages or infarcts may ensue, giving rise to a condition which may be known as hæmorrhagic cæcitis.

Perforations of the cæcum have occurred in cases of tuberculous or typhoid ulcerations, or cases of stercoral ulcers in patients who have been habitually constipated. Perforations have also occurred as result of foreign bodies which have penetrated this portion of the intestinal tract. Hagler,[5] in reporting his case of dissecting interstitial abscess of the cæcal wall, wrote: "It is remarkable that there should have been no adhesions or gangrene, and that the pus should have localized between the layers of the cæcal wall. It is interesting to speculate whether perforation would have occurred into the lumen of the cæcum or into the free peritoneal cavity if operation had been delayed." It is probable that in the case reported herein the patient had an abrasion of the mucosa by fæcal masses, and that an acute phlegmonous inflammation was added to this, just as similar inflammations have existed in the stomach or in the subcutaneous tissues in various parts of the body, and that this inflammatory process terminated in mesenteric thrombosis with resulting gangrene. Of the few cases reported in the literature of gangrene with perforation of the cæcum, one finds a case reported by Bowen[1] in a young lady of twenty-seven years who had a perforation of the cæcum near the ileocæcal valve associated with acute appendicitis. Dickinson[3] reported three cases of perforated ulcers of the cæcum, two of which died. Hallopeau and Monod[6] reported a fatal case of massive gangrene of the cæcum. Dowd[4] reported a case of resection of an acute suppurative inflammation of the colon, but this did not involve the cæcum.

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BOOK REVIEW

DIE CHIRURGIE DER TUBERKULOSE. By P. CLAIRMONT, O. WINTERSTEIN and A. DIMTZA. (Surgical University Clinic, Zürich), 8 vo.; pp. 661; 392 illustrations. Berlin, S. Karger, 1931.

A most comprehensive work presenting in one volume the surgical treatment of various forms of tuberculosis. As a corollary of their main thesis that a tuberculous lesion almost anywhere in the body may possibly be amenable to such treatment, the authors advocate dropping the term "surgical tuberculosis" with its usual narrow meaning. According to their view, there is no form of tuberculosis which belongs exclusively to the surgeon, nor is the surgeon wholly excluded from any part of the therapeutic domain of this disease. The book passes in review the numerous situations in which surgery may have a part, the text being supplemented by a generous number of excellent illustrations, some of them in color. Most of the recognized clinical forms of tuberculosis receive due attention. Some of the problems mentioned for surgical approach are rarities in practice, but the suggestions at least stimulate thought. In such instances the text sometimes represents merely a citation of literature. The experience of the authors, however, has obviously been wide and constitutes the background of much of their writing, especially in such sections as those on urogenital tuberculosis and tuberculosis of the bones and joints. An exception which will be disappointing to some readers is the section on tuberculosis of the lungs, by far the most frequent form of the disease encountered in actual experience. The advance of chest surgery has been rapid and brilliant in recent years. Collapse and immobilization of the diseased lung by artificial pneumothorax or by one of the more strictly surgical operations such as thoracoplasty is without question the most valuable adjunct of standard rest treatment, and the increasing use of these measures will go far not only to alleviate pulmonary tuberculosis but also to prevent the complications which so often owe their origin to the pulmonary focus. For these reasons the subject warrants more detailed discussion in such a book, although it is perhaps too much to expect that even the collective experience of three men could include so many highly specialized fields.

Great care is evident in the presentation of the modern conception of the pathogenesis and natural evolution of various tuberculous lesions, chief attention being given to the views of the German pathologists who have done such illuminating work in this branch. It is self-evident that no one should attempt to treat a chronic infectious disease like tuberculosis, especially by radical manipulation, unless he can rightfully lay claim to a knowledge of its probable behavior in terms of pathogenetic laws, yet there are some—usually labelled by their impetuosity—who violate this fundamental axiom to the

harm of the patient and the discredit of surgery. If the surgeon has not the time or interest to go into this very profoundly he should at least be guided by the counsel of the internist in possession of the information. Happily, in many centres there has sprung up a definite plan of coöperation between surgeons and internists in handling these cases, the consequence being more conservative treatment and better end-results. This book exemplifies the attitude of a clinic where sound comprehension of the pathogenesis and clinical course of tuberculosis dominates therapeutic action—certainly an ideal. One cannot read the paragraphs on tuberculous peritonitis, for instance, without appreciating the refined judgment which guides the authors and without doubting the wisdom of some surgeons in almost routinely advising laparotomy in such cases.

Surgical technic is not discussed in detail. The purpose of the book is evidently to help fill the need of providing a broad understanding of the disease, a requisite condition in successful surgery.

There is a useful chapter outlining methods of demonstrating tubercle bacilli in tissues and fluids and including a discussion of the blood picture and the tuberculin reaction.

A closing chapter is devoted to the Sauerbruch-Hermannsdorfer and the Gerson salt-poor diets, which the authors have tried in 145 cases. They report the diets to be of definite help in tuberculosis of the bones and joints and adjacent soft parts, but feel that diet alone should not be depended upon to the exclusion of heliotherapy, X-ray therapy and general rest treatment.

J. Burns Amberson, Jr.

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Remittances for Subscriptions and Advertising and all business communications should be addresed to the

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Philadelphia, Penna.